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BUREAU OF HYGIENE AND TROPICAL DISEASES.

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A REVIEW OF REFERENCES TO THE BONE LESIONS OF YAWS.*

By C. J. HACKETT, M.D., M.R.C.P., D.T.M. & H.

Director, Wellcome Museum of Medical Science.

There are comparatively few articles whose titles strictly refer to this subject, and still fewer are based upon the study of the radiographs of more than one or two cases. For purposes of discussion, the relevant publications fall conveniently into two groups. The first group includes papers in which bone lesions are mentioned in lists of cases that have received various treatments or are briefly referred to in descriptions of the disease as a whole ; the second group comprises papers dealing in more detail with the bone lesions, and sometimes including radiographical and pathological data. Seventy-four articles will be discussed, but some others that contained nothing original have been disregarded.

The First Group.

Of 40 papers falling into the first group, in 26 mention is made of bone lesions during the *secondary* stage of the disease, in 3 papers such an association is denied, but in 11 there are records of various bony lesions occurring during this stage.

HARPER (1917) Fiji, GUTIERREZ (1922) Philippines, and HUNT and JOHNSON (1923) Samoa, regard all bone lesions as tertiary even (GUTIERREZ) if secondary skin lesions are also present.

Of those who record bone lesions during the secondary stage, LENZ (1909) East Africa, speaks of bone and joint pain, and MOSS and BIGELOW (1922) Dominican Republic, mention swollen and painful joints. The following types of lesions were reported by the other authors :—

Periostitis : RAT (1891), Leeward Islands ; HALLENBERGER (1916), Cameroons ; ARAUJO (1928), Brazil ; HERMANS (1931), Dutch East Indies ; TODD (1931), Belgian Congo ; D'SOUZA (1933), Nicobar Islands.
Chronic Periostitis : VISWALINGAM (1922), Malaya.
Osteo-periostitis : ARAUJO (1928), Brazil ; TAKASAKI (1932), Caroline Islands.
Proliferative osteitis : HALLENBERGER (1916), Cameroons.
Nodes : CHAMBERS (1938), Jamaica.
Dactylitis : HALLENBERGER (1916), Cameroons ; VISWALINGAM (1922), Malaya ; CHAMBERS (1938), Jamaica.
Arthritis : HERMANS (1931), Dutch East Indies ; TAKASAKI (1932), Caroline Islands ; D'SOUZA (1933), Nicobar Islands.
Sabre-tibia : HALLENBERGER (1916), Cameroons ; VISWALINGAM (1922), Malaya.

* The material for this review was collected while holding a Senior Fellowship in Tropical Medicine, of the Medical Research Council, Great Britain.

The various bones were reported to be involved as follows :—

Skull and clavicle, each 3 times ; scapula, humerus, ulna, radius, and carpus, once each ; metacarpals and phalanges, 5 times ; ribs, once ; sternum, twice ; femur and patella, each once ; tibia, 4 times ; tarsus, once ; metatarsals, twice ; long bones, once.

The following references to *tertiary* bone lesions are made in the same 40 articles. Moss and Bigelow (1922), Dominican Republic, were not sure if tertiary bone lesions were due to yaws or to syphilis. Powell (1923), Assam, regards all descriptions of tertiary yaws lesions as mis-diagnoses for syphilis. He had seen no such yaws lesions develop among the population whom he had closely observed for ten years following the first introduction of yaws among them. Araujo (1928) Brazil, regards all the bone lesions he saw as secondary, because secondary skin lesions were present. He had never seen gummata or ulceration due to yaws, and does not accept a tertiary stage in yaws. Harley (1933), Liberia, does not state the stage of the disease in his cases.

Bone lesions were recorded by the following authors :—

Periostitis, acute, diffuse, localized, generalized, multiple or hypertrophic : Bahr (1914), Ceylon ; Harper (1914) (1917), Fiji ; Breinl (1915), Papua ; Moss & Bigelow (1922), Dominican Republic ; Hunt & Johnson (1923), Samoa ; Callanan (1925), Kenya ; Ramsay (1925), Assam ; Montel (1928), Cochin China ; Lambert (1929), South Pacific ; Dey (1930), Assam ; Wilson & Mathis (1930), Haiti ; Fitzgerald & Dey (1931), Assam ; Todd (1931), Belgian Congo ; Hermans (1931), Dutch East Indies ; Harley (1933), Liberia ; de Langen & Lichtenstein (1937), Dutch East Indies ; Manson-Bahr (1945) ; Strong (1944).

Chronic periostitis : Rat (1891), Leeward Islands ; Bahr (1914), Ceylon ; Gutierrez (1922), Philippines ; Callanan (1925), Kenya ; Manson-Bahr (1945).

Periostitis with ulceration : Baermann (1911), Sumatra ; Harper (1914), Fiji ; Breinl (1915), Papua ; Wilson (1924), Haiti ; Strong & Shattuck (1930), Liberia.

Periosteal nodes : Howard (1908), Nyasaland ; Gutierrez (1922), Philippines ; Manson-Bahr (1945) ; Strong (1944).

Nodes : Breinl (1915), Papua ; Howard (1915), East Africa ; Dey (1930), Assam ; d'Souza (1933), Nicobar Islands.

Nodes with ulceration : Howard (1908), Nyasaland ; Lambert (1929), South Pacific.

Bone lesions with ulceration : Chambers (1938), Jamaica.

Osteitis, hypertrophic, diffuse, localized or multiple : Bahr (1914), Ceylon ; Wilson (1924), Haiti ; Buxton (1928), Oceania ; Montel (1928), Cochin China ; Wilson & Mathis (1930), Haiti ; Hermans (1931), Dutch East Indies ; de Langen & Lichtenstein (1937), Dutch East Indies ; Serra (1938), Belgian Congo ; Manson-Bahr (1945).

Gummata : Lenz (1909), East Africa ; Viswalingam (1922), Malaya ; Montel (1928), Cochin China ; Takasaki (1932), Caroline Islands ; de Langen & Lichtenstein (1937), Dutch East Indies.

Gummatous periostitis : Baermann (1911), Sumatra ; Hermans (1931), Dutch East Indies.

Gummatous nodes : Dey (1930), Assam.

Gummatous osteitis : Baermann (1911), Sumatra ; de Langen & Lichtenstein (1937), Dutch East Indies.

Periosteal gummata with ulceration : Rat (1891), Leeward Islands : Hallenberger (1916), Cameroons ; Hermans (1931), Dutch East Indies ; Takasaki (1932), Caroline Islands ; de Langen & Lichtenstein (1937), Dutch East Indies.

Dactylitis: RAT (1891), Leeward Islands; CASTELLANI (1907), Ceylon; LENZ (1909), East Africa; BREINL (1915), Papua; DE BOISSIERE (1917), Fiji; HARPER (1917), Fiji; MOSS & BIGELOW (1922), Dominican Republic; CALLANAN (1925), Kenya; BUXTON (1928), Oceania; DEY (1930), Assam; STRONG & SHATTUCK (1930), Liberia; WILSON & MATHIS (1930), Haiti; TODD (1931), Belgian Congo; HARLEY (1933), Liberia; DE LANGEN & LICHTENSTEIN (1937), Dutch East Indies; SERRA (1938), Belgian Congo; MANSON-BAHR (1945); STRONG (1944); MACKIE *et al.* (1945).

Sabre-tibia: CASTELLANI (1907), Ceylon; BAHR (1914), Ceylon; WILSON (1924), Haiti; BUXTON (1928), Oceania; DEY (1930), Assam; WILSON & MATHIS (1930), Haiti; HERMANS (1931), Dutch East Indies; TAKASAKI (1932), Caroline Islands; D'SOUZA (1933), Brazil; HARLEY (1933), Liberia; DE LANGEN & LICHTENSTEIN (1937), Dutch East Indies; SERRA (1938), Belgian Congo; MANSON-BAHR (1940); STRONG (1944); MACKIE *et al.* (1945).

Epiphysitis: WILSON & MATHIS (1930), Haiti; MANSON-BAHR (1945).

Spontaneous fracture: MANSON-BAHR (1945).

The various bones were reported to be involved as follows:—

Skull, 11 times; clavicles, 10 times; scapula and humerus, each once; ulna and radius, each 12 times; metacarpals and phalanges, 7 times; hand bones, twice; ribs, 7 times; sternum, 10 times; vertebrae, once; femur, twice; tibia, 18 times; fibula, twice; metatarsals, once; foot bones, once; limb bones, 7 times.

The foregoing summaries may be amplified by the following notes:—

BAHR (1915), Ceylon, observed hard bony nodules resulting from the subsidence of periostitis and diffuse osteitis, leading to sabre-tibia and often bowing of the forearms. "Apparently a rarefying process occurs in the centre of long bones leading to spontaneous fracture and mal-union." Epiphysitis of phalanges and metacarpals leading to deformity and shortening of digits was noted and "one case of ulceration obviously associated with bone lesions". HARPER (1914), Fiji, speaks of perichondritis of the costal cartilages; the same author (1917), Fiji, records that 15 per cent. of 542 tertiary cases had bone lesions, and that 75 per cent. of these involved the lower limb. BREINL (1915), Papua, apparently reports only tertiary yaws bone lesions, and would regard some of them as a separate disease. Illustrations depict absorption of phalanges and ulceration of legs, probably related to bone lesions. HOWARD (1915), East Africa, reports that in children, stunting or overgrowth of the ulna may lead to dislocation of the hand. GUTIERREZ (1922), Philippines, found sub-periosteal nodes which altered the size and shape of the bone; periostitis was present in over 40 per cent. of cases of tertiary yaws. WILSON (1924) Haiti, saw the femur affected only once, and thought the humerus, ribs and vertebrae were exempt.

BUXTON (1928), Samoa, observed ulceration with osteitis. He regarded bone lesions as tertiary, for he saw few cases of primary or secondary yaws since recent systematic mass treatment had been moderately effective. MONTEL (1928), Cochin China, observed ossifying periostitis with eburnation, but more often he found such destructive processes as osteo-chondritis, osteoporosis with rarefaction of osseous trabeculae, or rarefying osteitis; purely localized gummatous forms led to bony destruction and punched-out perforations of bones which were well seen in radiographs. He thought tertiary yaws lesions less frequent and more benign than tertiary syphilitic lesions. LAMBERT (1929), South Pacific, says that if periosteal nodes occur near the middle of a bone they cause bending; if at the ends, in young people, they lead to lengthening of the bone. Necrosis of bone is, in his experience, rare. In 1423 cases, WILSON and MATHIS (1930), Haiti, found rheumatic pain in 46·8 per cent., painful swollen joints in 4·7 per cent., periostitis and osteitis in 4·7 per cent., dactylitis in 0·5 per cent., sabre-tibia in 0·4 per cent.

DEY (1930) reported deformity of fingers and shortening of phalanges. STRONG and SHATTUCK (1930), Liberia, report dactylitis with or without ulceration and bony destruction. TODD (1931), Belgian Congo, says that the periostitis of the secondary stage may accompany or precede the generalized skin eruption. TAKASAKI (1932), Caroline Islands, speaks of diffuse or circumscribed thickenings of the tibia and other bones, immediately beneath the skin. HARLEY (1933), Liberia, found bone lesions in 28 per cent., and rheumatic pain in 56 per cent., of 5,597 yaws cases ; he also observed bowing of the forearm bones. He concludes that there are apparently two types of advanced active yaws, one characterized by ulceration of the skin and soft parts, and the other by rheumatic pain and involvement of the bones. DE LANGEN and LICHTENSTEIN (1937), Dutch East Indies, speak of a diffuse osteitis and periostitis, especially of the tibia, leading to thickening and bowing, and thus to sabre-tibia. SERRA (1938), Belgian Congo, says that tertiary yaws bone lesions resemble those of syphilis, and that the general health is usually good. He also noted irregular thickening of the tibia and hypertrophy of the femoral condyles. Parts exposed to frequent injuries were most often affected. FOX (1944) speaks of osteoperiostitis of the long bones especially the tibia, ulna, fingers and clavicle, and synovitis and tenosynovitis as occurring in the tertiary stage. He does not mention secondary bone lesions.

The Second Group.

The 34 remaining articles to be discussed deal with the bone lesions of yaws in more detail, and many of them are based on some radiological or pathological data. With one or two exceptions they will be dealt with in chronological order.

SCHÜFFNER (1907), Sumatra, speaks of bone and joint pains, worse at night, which were sometimes marked in the first six months of the disease ; adults suffered more than children. Although these pains were present in 20 per cent. of cases, he found no radiographical changes. They occurred in the first eruption or during relapses, but later in the disease they were less evident. They could not be differentiated from the pains of syphilis, except that they were more severe and frequent. Mercury was most effective in their treatment. He found thickening of the bones of the extremities, without spontaneous pain, in 4 of 63 adults and in 13 of 66 children under 14 years of age ; all these 13 had the typical generalized yaws skin eruption. He reports periostitis of the middle and index fingers, fifth metatarsal, ulna and fibula, causing swelling of the part. He found no rarefaction of bone tissue although his illustrations show rarefied foci in the periosteal deposits. On healing, the affected bones returned to normal. He claims that the periostitis is like that of syphilis, but that its localization to only part of a long bone, the absence of pain, and its onset in children, allow of certain differentiation. These changes are not related to gummata of bones and respond well to mercury.

BAERMANN and SCHÜFFNER (1912), Dutch East Indies, regard the periostitis of the early stage of yaws as characteristic and especially frequent in children. They point out that even in the early stage of the disease, lesions are not confined to the skin. In the tertiary stage there may be enormous osteitic and periostitic thickening of the tibia and bone lesions with gummatous-cystic formation.

A paper by MAUL (1918), Philippines, is frequently and uncritically quoted. He describes 10 out of 20 cases with bone lesions found in 100 cases of yaws. The initial diagnosis of bone lesions in these cases was apparently based upon the complaint of pain. He publishes illustrations of only 4 of the cases. The radiographs reproduced of his "type" case are poor, and suggest periosteal deposits along the radius and ulna which are not in his descriptions. Secondary

yaws skin lesions were present only in his "type" case, a woman aged 27 with yaws of 5 months' duration; in the other three cases the skin lesions had healed 9 months to 5 years previously. One of these patients, aged 15, had contractures and large ulcers, and the radiographs resemble what other writers would describe as early gummata. In another patient, aged 15, he illustrates small areas of rarefaction in the articular surface of the tibial epiphysis, and cortical thickening, periosteal deposits and small cortical rarefied foci of the phalanges, with some destruction. In the fourth case there was one small cortical rarefied focus in the calcaneum. The illustrations of Maul's type case show well-defined large oval foci, up to 2–3 cm. in length, of cortical rarefaction in and on the surface of the cortex of the radius and ulna, with probably little or no periosteal reaction. Maul says that "most of the lesions appear to originate in the interior of the bone", but this cannot be determined from the illustrations. In the hand bones are shown small cortical rarefied foci and some periosteal deposits, which apparently resolved with treatment. The nodular lesions which he reports in 2 per cent. of cases, and the chronic lesions which he says differ from the earlier ones, are not mentioned in his case notes. His illustration of "chronic bone lesions with deformity" is probably of lesions of tertiary yaws, but he gives no details of the case. He differentiates yaws bone lesions (excluding the 2 per cent. nodular ones) from syphilitic by the absence of both periosteal proliferation and cortical thickening in the former. In the nodular lesions the thickening remains localized, and sooner or later shows central rarefactions, thus becoming the periosteal gummata of other authors. In Maul's 20 cases the frequency of involvement of bones was as follows:—tibia, 8; fibula, 7; calcaneum, and phalanges of hands and feet, each 6; metacarpals and radius, each 4; humerus and patella, each 3; ulna, femur and tarsus, each 2; carpus, ribs, scapula, sternum and pelvis, each once. Lesions of vertebrae and clavicle were not observed. He concludes that in his series the joint pains were "due in the most part to the presence of lesions of the articular surfaces", although of his 20 cases 7 had only 2 lesions, and 5 only 1 lesion in the whole skeleton. He concludes that "regeneration of the bone is complete at the site of the lesion, if the destruction has not been too great."

Fernandez (1917), Philippines, at the meeting when Maul (1918) read his paper, showed a radiograph of yaws lesions of the vertebrae, but no illustration is published.

van Nitsen (1920), Northern Rhodesia, says that osteocopic pains occur in the secondary and tertiary stages. Of 699 yaws cases, 459 had tertiary yaws lesions, among which the following bone lesions were observed; osteitis and periostitis including thickened long bones, sabre-tibiae, exostoses and dactylitis (77); bony necrosis from extension of yaws ulcers (27); bony rarefaction and absorption of phalanges (3). Van Nitsen does not mention gummatous lesions, although his cases of bony necrosis were probably of this nature. He says that tertiary lesions may occur at any age, even in infants of 18 months, and that the tertiary period starts towards the end of the secondary stage, either with a relapse of the eruption or 10–30 years after the secondary lesions have healed.

Clapier (1921), French Equatorial Africa, reports that the hypertrophic osteitis of yaws appears in children when the secondary rash has been generalized for some months, often when it is subsiding; but some typical yaws are usually still present. The principal lesions are: (1) goundou; (2) osteitis of the short bones, *e.g.* scapula, trapezium, scaphoid, etc.; (3) osteitis of the epiphyses of long bones; (4) osteitis of the diaphysis of long bones, with changes in length, thickness and form which may be either localized or generalized (sabre-tibia). The phalanges are often swollen. These changes are rarely single, are usually bilateral, and are always accompanied by simple muscular

atrophy. In the Oubangui, Clapier found that in 10 per cent. of 600 yaws cases there were bone lesions. These became less marked as the bony system developed, but permanent deformities resulted in half the cases. He rules out rickets and congenital syphilis as causative factors. He regards yaws osteitis as a tertiary lesion which nearly always encroaches on, or prolongs, the secondary stage.

SPITTEL (1923), Ceylon, describes periosteal nodes and bony tenderness in the secondary stage but says they " belong more appropriately to the tertiary period." As tertiary bone lesions, he describes localized gummata or nodes which may ulcerate, and diffuse periostitis and osteitis leading to osteosclerosis with bowing and deformity of long bones (sabre-tibia). Central gummata of the phalanges, and absorption of these bones, with shortening and ankylosis, may also occur. No bone is exempt, but sites of muscular attachments are particularly affected. SPITTEL found the vertebrae involved in one case.

GUERRERO, FERNANDEZ and ROSAL (1924), Philippines, describe the case of a girl, aged 13, who had had yaws for 6 months. The radiographs showed cortical thickening and small foci of cortical rarefaction in the proximal phalanges and metacarpals, which healed under treatment with sodium-potassium tartro-bismuthate.

MAYER (1924) quotes SCHÜFFNER's description of the early bone lesions of yaws, and says that the later lesions consist of large osteitic and periostitic thickenings of the tibia with deformity, and also bony craters with gummatous cystic changes.

PLEHN and MENSE (1924) state that painful periostitic rarefactions, sometimes combined with proliferating osteitis, occur in the secondary stage. In the tertiary stage gummata on the sternum near the clavicle are characteristic. Periostitis and osteitis, sometimes with gummatous ulceration of the soft tissues, are also seen, and in children may lead to gross deformity.

BOTREAU-ROUSSEL and CORNIL (1924) describe the histopathological changes in specimens of goundou. These consisted of thickening of the periosteum and hypertrophy of the spongy tissue, with increased trabeculae. There were also fibrosis and perivascular plasma-cell collections in the spongy tissue. These were comparable with certain syphilitic changes and indicate that in yaws, as in syphilis, the bone marrow is particularly liable to attack.

BOTREAU-ROUSSEL (1925), Ivory Coast, found that 75 per cent. of 130 cases of yaws osteitis occurred during or soon after the generalized skin eruption. Of these 130 cases, goundou was present in 121, and the tibia was involved 69 times; the fibula, femur, radius, ulna, clavicle, humerus and phalanges were involved one to five times each. He describes cases of goundou with thickening of phalanges and long bones, and the development of sabre-tibiae during or soon after the yaws eruption. In this group of 130 cases, the bone lesions appeared while the secondary eruption was present in 49 per cent. (64), and soon after the eruption had healed in 33 per cent. (43). He regards these as tertiary lesions occurring during the secondary stage (see his Fig. 1). In the long bones hypertrophic osteitis was present. The periosteum was thick and adherent to the bone, the surface of which was very rough and irregular, owing to the enormous dilatation of the Haversian canals, whose external openings were up to 2 mm. in diameter, giving the bone a spongy appearance. The medullary canal of a sabre-tibia on section was completely filled with irregularly alveolar tissue; transversely, the bone was circular. Anterior bowing of the femora was also seen.

SOETOMO and EICHHORN (1925), Dutch East Indies, say that except for periostitis, most of the bone lesions of yaws are tertiary and resemble those of tertiary syphilis in the symmetry of their occurrence and in the sites of predilection—*e.g.* the occurrence of gummata in the tibia, forearm bones and cranium.

They point out that there is a fairly constant bony rarefaction surrounding the yaws lesion, so that often there is gummatous softening with hyperostosis and sclerosis surrounded by atrophic changes. The bony rarefaction is apparently connected with neurotrophic disturbances, and the resulting diminished rigidity probably leads to the deformities so often seen in framboesia, particularly in growing bones. The atrophy remains for some time after the healing of the yaws process. There is no bony atrophy in syphilis. Gummatous osteomyelitis, a rarity in syphilis, is not infrequent in yaws, and the cortex and periosteum show little reaction, so that spontaneous fractures or total necrosis may occur. The final diagnosis of tertiary yaws bone lesions may depend on the history, symptoms and locality. The Wassermann reaction is usually strongly positive and is rarely changed by treatment with salvarsan and mercury. The illustrations to this paper show large foci of rarefaction with localized periosteal deposits, sabre-tibia, a spontaneous fracture, indefinite cortical rarefied foci in phalanges, calcaneal changes and total necrosis of the humerus following gummata.

Knaggs (1926) states that in yaws, as in syphilis, the bones are affected in the tertiary stage. He quotes Maul (1918) and says that Maul's illustrations do not resemble syphilitic lesions. He describes a tibia from New Guinea (in the Royal College of Surgeons' Museum, England) which he compares with the lesions Maul describes, and he suggests that the lesions present were probably associated with ulceration. The pitted condition he regards as quite different from the usual appearances of syphilis of long bones. Other authorities would regard these lesions as multiple gummata.

Hashikuchi (1927) describes the cases of 5 children who had yaws two or more years previously and were incompletely cured. They all had positive Wassermann reactions even after the bone lesions had cleared up under treatment. All had dactylitis, and in 4 cases the long bones were affected. The terminal phalanges of the hands and the middle and terminal phalanges of the feet were not affected. One patient had slight nocturnal pain. Histo-pathological changes in material from the biopsy of one phalanx are reported. The periosteum was thickened and oedematous, with slight diffuse cellular infiltration of lymphocytes, plasma cells, fibroblasts and polymorphonuclear leucocytes near dilated small veins (*sic*). Indeterminate vascular changes were present. No necrotic or cicatricial changes and no spirochaetes were seen.

Polak (1927), Java, says that the bony changes of yaws occur chiefly in the tertiary stage, but they may appear early, even in children, and their differentiation from congenital syphilis is difficult. Periostitis and osteitis are the most frequent changes. Long and short tubular bones may be affected, but the tibia is most frequently attacked, often resulting in sabre-tibia. He describes the case of a boy, aged 15, who had yaws in infancy, and when seen had tendon contractures, ankyloses, tertiary ulcers, osteomyelitic foci with ulceration and spontaneous fractures, and sabre tibiae. The Wassermann reaction was positive and the active lesions responded quickly to neosalvarsan. He found that the X-ray pictures resembled those of tertiary syphilis. Symmetrical gummatous processes were found in the tibia, ulna, and radius, and the carpal and tarsal bones; the bones of the fingers and toes were also affected. In favour of yaws was the severe bony rarefaction, gummatous and atrophic, and the scanty cortical and periosteal proliferation. Cachexia is less frequent, and the viscera are very rarely attacked in yaws. The history and clinical findings assist in the diagnosis of yaws.

Schoebl (1928), Philippines, writes "it is an open question whether or not the bone lesions in yaws, either early or late, are always due to actual localisation of *Treponema pertenue* in the bones, or whether it is a process analogous to that of keratoderma plantare," which, he found, occurred in monkeys in a

state of partial immunity. He says that bone lesions should be classified by themselves since they are either early or late, hypertrophic, atrophic or necrotic, and may affect the periosteum or bone. In a private communication (1935) he says that in nearly 500 monkeys experimentally infected with yaws he found bone lesions developed only as an extension from skin lesions.

FOX (1929), Haiti, speaks of unilateral osteo-periostitis with fungating cutaneous gummata of the leg, indistinguishable from those due to syphilis. He illustrates an inactive lesion consisting of a dense localized cortical thickening on the anterior surface of a bowed tibia.

HASSELMANN (1931), Philippines, who had worked with SCHOEBL, says rarefying osteitis and periostitis, although claimed by clinical observers as an early manifestation of yaws, have never been observed in experimental infections in man or monkeys. Neither pathological changes typical of yaws nor *Treponema pertenue* have been found, and it has yet to be proved that the bone lesions are not an extension of a previously healed skin lesion.

MONTEL and COUPUT (1932), Indo-China, do not mention bone lesions during the secondary stage. They found osteo-periostitis in 10–20 per cent. of cases of tertiary yaws. It was most frequent in children and adolescents and in conditions of malnutrition. It appeared 3–6 years after the secondary eruption. The limb bones were usually affected, but the ribs, clavicles, and all other bones might be involved. Clinically these authors recognize two forms :—(1) localized nodular lesions with gumma-like " géodes " ; (2) diffuse osteitis, due either to extension of the nodular lesions into the cavity of the bone or to the original involvement of the cavity, which leads to destruction of the periosteum, to hypertrophy, or, more often, to rarefaction of the cortex and trabeculae, and also to osteoporosis. Ulceration, they say, may occur in both types. The lesions develop slowly without marked general symptoms except profound osteocopic pain, but cachexia may result. Arthritis, ankylosis and sabre-tibia may occur. Response to neoarsphenamine is good. Differentiation from syphilis is assisted by the presence of yaws in the community and by the history of the patient. Syphilitic bone lesions are probably never so generalized as yaws bone lesions, and in the latter the periosteal proliferation and cortical thickening so characteristic of syphilis are lacking. In 30 years in Cochin China Montel and Couput saw only one case of goundou, and in Indo-China, where yaws is prevalent, they saw none. They note the resemblance between the limb bone lesions described by BOTREAU-ROUSSEL (1925) and those they have seen in yaws cases. They suggest that goundou is a special aspect of African yaws, and gangosa of Philippine and Malayan yaws. The illustrations show large foci of cortical rarefaction, either isolated or in groups, with bony expansion and some periosteal deposition. In the phalanges are small foci with cortical thickening. The osteoporosis and trabecular changes are not well shown.

BEITZKE (1934) says that differentiation of yaws from syphilitic bone lesions is difficult. In yaws osteoporosis is usually present and periostitis is found during the secondary stage. Tertiary bone lesions may occur early, even while secondary skin lesions are still present ; they are periosteal and osteomyelitic gummata, which are usually localized, though numerous foci may exist in one bone. These lesions produce only slight bone formation. Spontaneous fractures are more frequent than in syphilis, and callus formation is slight. Periosteal and cortical gummata frequently ulcerate. The same bones are affected as in syphilis, the skull and tibia most frequently, but also the phalanges metacarpals, metatarsals, ulna and fibula. In the tibia, hyperostoses with sabre-tibia also occur. Sabre-tibia is more frequent in yaws than in syphilis. Contractures and ankyloses are also found.

MONTEL, MASSARI and LE-VAN-PHUNG (1934), Annam, describe the case of a boy, aged 15, who had yaws 9 years previously, with bone lesions of several years' duration. The affected parts were swollen and on the legs gummatous ulceration extended to the bone. Two types of lesion were seen in the radiographs; (1) localized diaphyseal thickenings with condensation of the periosteum and deep tissue, encircling the bone or on one side only; and (2) "géodes" [gummata] of various sizes, but always round and well-defined in these thickenings, and associated with further marked condensation. Between these last lesions there might be slight decalcification. The authors say that the number of the "géodes", the irregularity of the hyperostoses, and the narrowing of the medullary canal, differentiate these yaws lesions from those of syphilis. The illustrations are good and show expansion, cortical thickening and large foci of cortical rarefaction in the bones of the forearm.

WOLTER (1934) concludes from the absence of spirochaetes in them, that the lesions of the phalanges in yaws are an allergic reaction to the organism. He also suggests that the absence of spirochaetes in these lesions may be due to framboesial spores, such as have been assumed in syphilis, but he admits that this does not account for the predilection for the phalanges.

The *Jamaica Yaws Commission Report* for 1936 states that there are probably some bone lesions in the majority of cases of yaws in the early stages, with pain and tenderness. Perhaps 15 per cent. of yaws cases have bony swelling and tenderness. The long bones, especially of the forearm and leg, are most frequently affected, but the skull, phalanges, or any other bone may be involved. Bone changes are perhaps most frequent during the secondary stage, but they also occur later and may be associated with ulceration. The course of localized swelling, fluctuation and ulceration has been observed. Great deformity frequently results, and also bowing of the legs and forearms. The radiographical appearances are fairly constant and often characteristic: (1) widespread multiple areas of rarefaction, with or without surrounding increased density; and (2) fusiform enlargement due to periostitis causing thickening of the cortex; or (3) great enlargement with sclerosis of cortex and medulla. The Report for 1932 states that no spirochaetes were found in material collected from bone lesions.

HACKETT (1936), Australia, traces part of the development of sabre-tibia and describes certain other lesions found in aboriginal bones, which, he suggests, are due to yaws. No account was taken of the stages of the disease in which the lesions occurred. Secondary skin lesions were rarely seen. All the lesions that were suggested to be of yaws origin have been described by observers in yaws communities.

MACKAY (1938) criticizes the suggestion of HACKETT (1936) that certain lesions in aboriginal bones are due to yaws. He describes and illustrates a number of the 350 specimens he examined in the Australian Institute of Anatomy, Canberra, and divides the bony changes he observed into periostitis, osteitis, with rarefaction and sclerosis, and osteomyelitis. He concludes that they result from venereal syphilis, an endemic type of non-venereal syphilis, or some unknown indigenous disease. He regards venereal syphilis as the most probable. [Excluding the fractures and joint and skull changes, many other lesions strikingly resemble the bone lesions of yaws reported in yaws communities, and observed by Hackett in Uganda. MACKAY's exclusion of yaws as a possible cause is based upon published accounts of disease in Australian aborigines, without apparently, any observations or enquiries by himself concerning the diseases at present occurring in isolated aboriginal communities. The only articles on yaws bone lesions to which he refers are SPITTEL (1923), KNAGGS (1926) and P. MANSON "Tropical Diseases" (no date).]

BOTREAU-ROUSSEL, FRAGES and GAUTHIER-VILLARS (1937), Ivory Coast, describe the histopathology of yaws bone lesions in specimens from 20 biopsies. The main changes were thickening of the periosteum, in which were small collections of lymphocytes and plasma cells. They also observed vascular sclerosis, decalcification, laminar absorption and osteoclasis. There were increased fibrous tissue and plasma cell infiltration in the medullary spaces. Giant cells, marked bony destruction and spirochaetes were not found. These authors could not describe any changes as characteristic.

BOTREAU-ROUSSEL (1937), Ivory Coast, regards all the bone lesions of yaws as secondary lesions. Pain is present in most cases of secondary yaws at some time. Clinically there is no difference between the bone lesions (osteoperiostitis) at the onset and the deformities found years later. He never saw necrotic excavated lesions or gummatous nodules with ulceration such as those described by MONTEL in Indo-China. The onset, he says, may be abrupt or gradual with swelling over the bone, tenderness and increase of the pain, which is worse at night. General symptoms may be absent. Over 40 per cent. of 75 cases under 2 years of age had obvious bone lesions. In 130 cases the following bones were affected :—phalanges, 36 per cent. ; metacarpals and ulna each 15 per cent. ; tibia, 14 per cent. ; radius, 12 per cent. ; fibula, 5·5 per cent. ; femur, humerus and others, each 0·5 per cent. ; skull, 0·2 per cent. ; foot bones and goundou each 0·15 per cent. BOTREAU-ROUSSEL states that fingers may be swollen and turgid, the long bones thickened or fusiform and, in advanced cases deformed by exaggeration of normal curves or by exostoses. Severe pain may last months or years, but osteitis may continue after pain has ceased. In the hand, joints may be involved and spontaneous fractures occur, with resulting deformity. Ankyloses, mal-union of spontaneous fractures, and sabre-tibia may result. Diffuse or localized swellings of the skull are found. Soft tissue changes resolve and leave the bony thickenings more obvious. The histopathological changes are those he described earlier (1924 and 1937). In early cases the periosteum is thickened and the bone soft ; the bone is spongy from dilatation of the Haversian canals which, in marked cases, are filled with fibrous tissue. In old cases the cortex is dense and hard. In radiographs the periosteum is seen to be loosened and separated by inflammatory tissue ; this phase is soon followed by thickening of the underlying cortex and reaction of the medullary tissue, so that fusiform thickening of the bone and narrowing of the medulla result. In advanced lesions the Haversian canals, considerably enlarged by fibrous tissue, give the swollen bones the lacunar appearance of fibrous osteitis. Later there is organization, and bone is deposited round the enlarged Haversian canals. This author discards the suggestion that poor resistance or malnutrition plays an essential part in the production of bone lesions. His illustrations show sabre-tibiae, periosteal deposits, expansion and cortical thickening and small foci of cortical rarefaction, which he attributes to dilated Haversian canals.

CARTRON (1937), French Colonies, says the transition from the secondary to the tertiary stages is more theoretical than real. During the secondary stage rheumatic, joint and osteocopic pains, periostitis and dactylitis, occur; and in the tertiary, osteoperiostitis, bony deformity, contractures and ankylosis. These tertiary lesions may appear while the secondary lesions are still present, but more often they develop 2–6 years after infection, or even after a latent period of 30 years. It is difficult to differentiate tertiary yaws from syphilitic lesions, and figures for their incidence are of doubtful value as there is so often confusion. Cartron refers to MONTEL, COUPUT and BOTREAU-ROUSSEL for descriptions of yaws bone lesions. The incidence of tertiary yaws lesions is higher where the incidence of yaws is highest ; hence he is prepared to consider a tertiary stage in yaws and not to regard these lesions as syphilitic as he did until recently.

SIMPSON (1938), Cameroons, does not mention bone lesions during secondary yaws. Tertiary bone lesions commence five or more years after the secondary stage. The onset is indicated by pain and a small tender swelling. Radiographs show the periosteum raised from the cortex. This process extends along the bone. Decalcification of the underlying cortex follows, causing a " moth-eaten " appearance and calcification of the periosteal changes takes place ; finally, diminished vascular supply leads to bony sclerosis. Ankylosis and ulceration may occur. The apparent bending of the tibia, according to this author, is due to periosteal deposits on the anterior surface and not to deformity of the long axis of the shaft. There were no cases of sabre-tibia in 920 cases of yaws seen by himself, nor in the 30,000 yaws cases dealt with by 80 African yaws attendants. He suggests that some dietetic deficiency is the cause of this deformity. He says " the whole of the pathology of yaws is confined to the surface of the body, the skin and superficial bones being the chief sites of the lesions ", *e.g.* the leg, forearm and clavicle. Post-mortem examination of 20 cases of suspected yaws showed osteitis with varying degrees of rarefaction and sclerosis of bone. His illustrations, which are poor, show periosteal deposits, cortical thickening, and expansion and cortical rarefaction and periosteal deposits.

BRYANT and FAIRMAN (1939), Sudan, say that bone lesions in yaws are common in Nilotics (Dinka, Shilluk and Nuer) and uncommon in forest dwellers (Azanda, Bongo and others).

WILLIAMS (1939), in a recent text-book of X-ray diagnosis, quotes only from MAUL (1918), KNAGGS (1926) and WILSON and MATHIS (1930), and repeats the confusion of comparing the New Guinea tibia (R.C.S. England) with the changes described by MAUL.

GOLDMANN and SMITH (1943) from a study of 101 cases in African soldiers and civilians at Sierra Leone stress the difficulties of differential diagnosis.

Diagnosis of yaws was based upon the presence of typical yaws scars, quick response to treatment and the positive Kahn reaction. The following frequency of bone involvement was observed, the tibia in 46 cases, fibula in 20, femur in 13, ulna in 10, humerus in 9, radius in 7, and other bones each in less than 5. The authors illustrate a vertebral lesion and report skull lesions and spontaneous fractures. Gummata were noted, especially in the ribs, clavicle and pelvis. Trauma was considered an important factor in initiating lesions. During active growth joints and epiphyses are attacked.

The first sign of activity is said to occur in the cortex and resembles the lesions described by MAUL (1918) or are of more moth-eaten appearance. The later stages they regard as either heavy thickened bones or shrunken bent ones.

They believe the absence of periosteal deposits in the presence of cortical lesions [the bony expansion they described must have arisen from the periosteum] and the frequency of fibular lesions in yaws differentiate it from syphilis. Other lesions that might lead to confusion are osteomyelitis, osteitis deformans and tuberculosis. [It is probable that the authors refer only to tertiary bone lesions ; they do not mention any secondary bone lesions.]

HELFET (1944) deals with acute bone lesions in West Africans, mostly soldiers. In all cases the Wassermann and Kahn reactions were positive. He regards trauma as often initiating the lesions which soon develop marked changes. Acute onset with " rheumatic " pain and fever was frequent. The tibia, femur, clavicle and humerus were most frequently affected, but vertebral and skull lesions occurred. Joints and tendon sheaths were also involved. He says that yaws bone lesions are more rapid in development and are more painful than syphilitic ones.

X-ray appearances were increased diameter and density of bones or multiple layered periosteal deposits over diffuse cortical rarefactions. Microscopically

the bone lesions resembled syphilitic lesions except for the absence of endarteritis. [In this paper, too, probably only tertiary lesions are described.]

ASH and SPITZ (1945) regard bone lesions as tertiary. They say the tibia is most frequently involved and the early lesions are small foci of cortical rarefaction which lead to a diffuse worm-eaten appearance of the entire shaft. The resulting weakening of the bone leads to sabre-tibia. They speak of the development of " marble bones " in which fracture easily occurs. Gummata may occur in flat bones but are seldom in long bones. In children epiphyses and joints may be affected. The absence of periostitis in the presence of osteoporosis in yaws differentiates it from syphilis. They include goundou and gangosa in the tertiary stage. [Their illustrations of lesions of the hand bones are almost certainly secondary not tertiary.]

Summary.

The 70 articles dealt with above, although not including every reference to the bone lesions of yaws, are representative of the literature of this subject. The first point that comes to notice is the absence of any complete radiological study of these lesions. French and Dutch authors have added most to the knowledge of the subject since the earlier work of SCHÜFFNER (1907) and MAUL (1918).

Many of the differences in the various descriptions are probably due to the incompleteness of the studies upon which they are based, but it is remarkable that BOTREAU-ROUSSEL (1937) Ivory Coast, saw no destructive bone lesions comparable with those described by MONTEL *et al.* (1932 and 1934), Indo-China, SOETOMO and EICHHORN (1925) and POLAK (1927), Dutch East Indies, HACKETT (1936), Australia, and by others in other parts of the tropics, who had made no radiographs. Although these " bony gummata " were described in other parts of Africa by HOWARD (1908), Nyasaland, LENZ (1909), East Africa, HALLENBERGER (1916), Cameroons, and probably SIMPSON (1938), Cameroons, no radiological evidence has been produced.

Another feature of the literature is the absence, in many articles, of any reference to bone lesions occurring during the secondary stage of yaws ; while some authors maintain that all bone lesions belong to this period of the disease others state that they are all tertiary lesions. Here again there is no definite geographical grouping to account for the differences found. In the series of cases studied in Uganda by the reviewer, and to be reported elsewhere, lesions were found in all stages of yaws, and practically all the lesions described above were observed.

REFERENCES.

ARAUJO, O. da S. (1928) *Bull. Soc. Path. Exot.*, v. 21, 387. [*Trop. Dis. Bull.* 1929, v. 26, 169.]

ASH, J. E. & SPITZ, S. (1945) Pathology of Tropical Diseases. p. 50. Philadelphia and London : W. B. Saunders Company. [*Trop. Dis. Bull.* 1946, v. 43, 691.]

BAERMANN, G. (1911) *Arch. f. Schiffs- u. Trop.-Hyg.*, v. 15, Beiheft 6, 333.

—— & SCHÜFFNER, W. (1912) *ibid.*, v. 16, Beiheft 4, 327.

BAHR, P. (1915) *Ann. Trop. Med. & Parasit.*, v. 8, 675. [*Trop. Dis. Bull.* 1915, v. 5. 421.]

BEITZKE, H. (1934) *Handbuch spez. Path. Anat. u. Hist.*, v. 9, Pt. 2, 635. Berlin : Julius Springer.

DE BOISSIERE, R. (1917) *Edinburgh Med. J.*, n.s., v. 19, 226.

BOTREAU-ROUSSEL, J. M. (1925) Ostéites pianiques " Goundou ". Paris : Masson et Cie. [*Trop. Dis. Bull.* 1925, v. 22, 421.]

——. (1937) *J. Chir.* Paris, v. 50, 145.

—— & CORNIL. (1924) *Bull. Soc. Path. Exot.*, v. 17, 863. [*Trop. Dis. Bull.* 1925, v. 22, 651.]

——, FARGES & GAUTHIER-VILLARS. (1937) *Ann. d'Anat. Path. Méd.-Chir.*, v. 14, 699.

BREINL, A. (1915) *Ann. Trop. Med. & Parasit.*, v. 9, 285. [*Trop. Dis. Bull.* 1915, v. 6, 353.]

BRYANT, J. & FAIRMAN, H. D. (1939) *Lancet*, Apr. 22, 923.

BUXTON, P. A. (1928) Researches in Polynesia and Melanesia. Parts V–VII, 87. *No. 2 Mem. Series of London School of Hygiene and Tropical Medicine.* [*Trop. Dis. Bull.* 1929, v. 26, 436.]
CARTRON. (1937) *Bull. Office Internat. d'Hyg. Pub.*, v. 29, 541. [*Trop. Dis. Bull.* 1937, v. 34, 962.]
CALLANAN, J. C. (1925) *Trans. Roy. Soc. Trop. Med. & Hyg.*, v. 19, 312. [*Trop. Dis. Bull.* 1926, v. 23, 446.]
CASTELLANI, A. (1907) *Arch. f. Schiffs- u. Trop.-Hyg.*, v. 11, 19.
CHAMBERS, H. D. (1938) Yaws (Framboesia tropica). pp. 97, 105. London: J. & R. Churchill, Ltd. [*Trop. Dis. Bull.* 1939, v. 36, 88.]
CLAPIER. (1921) *Ann. Méd. et Pharm. Colon.*, v. 19, 75.
DEY, N. C. (1930) *Indian Med. Gaz.*, v. 65, 421. [*Trop. Dis. Bull.* 1931, v. 28, 450.]
FERNANDEZ, R. (1917) *Philippine J. Sci.* Sect. B, v. 12, 260.
FITZGERALD, G. H. & DEY, N. C. (1931) *Indian Med. Gaz.*, v. 66, 425. [*Trop. Dis. Bull.* 1932, v. 29, 385.]
FOX, H. (1929) *Arch. Dermat. & Syph.*, v. 20, 820. [*Trop. Dis. Bull.* 1930, v. 27, 713.]
——. (1944) Bercovitz' Clinical Tropical Medicine. p. 305. New York and London: Paul B. Hoeber, Inc. [*Trop. Dis. Bull.* 1944, v. 41, 787.]
GOLDMANN, C. H. & SMITH, S. J. (1943) *Brit. J. Radiology*, v. 16, 234. [*Trop. Dis. Bull.* 1943, v. 40, 919.]
GUERRERO, L. E., FERNANDEZ, R. & ROSAL, I. (1924) *Far-Eastern Ass. Trop. Med. Trans. 5th Bienn. Congr. Singapore, 1923*, p. 578. London: John Bale, Sons & Danielsson, Ltd. [*Trop. Dis. Bull.* 1925, v. 22, 550.]
GUTIERREZ, P. D. (1922) *Arch. Dermat. & Syph.*, v. 6, 265. [*Trop. Dis. Bull.* 1923, v. 20, 175.]
HACKETT, C. J. (1936) Boomerang Leg and Yaws in Australian Aborigines. London: Roy. Soc. Trop. Med. & Hyg., Monogr. 1.
HALLENBERGER. (1916) *Arch. f. Schiffs- u. Trop.-Hyg.*, v. 20, Beihelft 3, 153.
HARLEY, G. W. (1933) *J. Trop. Med. & Hyg.*, v. 36, 217; 235; 252. [*Trop. Dis. Bull.* 1934, v. 31, 272.]
HARPER, P. (1914) *Lancet*, Aug. 8, 370. [*Trop. Dis. Bull.* 1914, v. 4, 497.]
——. (1917) *Trans. Soc. Trop. Med. & Hyg.*, v. 10, 82. [*Trop. Dis. Bull.* 1917, v. 10, 82.]
HASHIKUCHI, M. (1927) *Acta Derm.*, Kyoto, v. 10, 616.
HASSELMANN, C. M. (1931) *China Med. J.*, v. 45, 1131. [*Trop. Dis. Bull.* 1932, v. 29, 388.]
HELFET, A. J. (1944) *J. Bone & Joint Surgery*, v. 26, 672. [*Trop. Dis. Bull.* 1945, v. 42, 387.]
HERMANS, E. H. (1931) *Acta Leidensia* (*Scholae Med. Tropicae*), v. 6, 64, 82.
HOWARD, R. (1908) *J. Trop. Med. & Hyg.*, v. 11, 197.
——. (1915) *ibid.*, v. 18, 25. [*Trop. Dis. Bull.* 1915, v. 5, 420.]
HUNT, D. & JOHNSON, A. L. (1923) *U.S. Nav. Med. Bull.*, v. 18, 599. [*Trop. Dis. Bull.* 1923, v. 20, 840.]
JAMAICA YAWS COMMISSION REPORT FOR 1936. (1936) p. 3. Kingston: Government Printing Office. [*Trop. Dis. Bull.* 1937, v. 34, 959.]
KNAGGS, R. L. (1926) The Inflammatory and Toxic Diseases of Bone. p. 126. Bristol: John Wright & Sons, Ltd.
LAMBERT, S. M. (1929) *Amer. J. Trop. Med.*, v. 9, 429. [*Trop. Dis. Bull.* 1930, v. 27, 710.]
DE LANGEN, C. D. & LICHTENSTEIN, A. (1936) A Clinical Text-Book of Tropical Medicine. p. 110. Batavia: G. Kolff & Co. [*Trop. Dis. Bull.* 1936, v. 33, 161.]
LENZ. (1909) *Arch. f. Schiffs- u. Trop.-Hyg.*, v. 13, 345.
MACKAY, C. V. (1938) *Med. J. Australia*, v. 2, 537.
MACKIE, T. T., HUNTER, G. W. & WORTH, C. B. (1945) A Manual of Tropical Medicine. p. 73. Philadelphia and London: W. B. Saunders Company. [*Trop. Dis. Bull.* 1945, v. 42, 665.]
MANSON-BAHR, P. H. (1945) Manson's Tropical Diseases. 12th edit. p. 596. London: Cassell & Co., Ltd. [*Trop. Dis. Bull.* 1946, v. 43, 607.]
MAUL, H. G. (1918) *Philippine J. Sci.*, Sect. B, v. 13, 63.
MAYER, M. (1924) Exotische Krankheiten. p. 94. Berlin: Julius Springer. [*Trop. Dis. Bull.* 1924, v. 21, 1002.]
MONTEL, L. R. (1928) *Bull. Soc. Path. Exot.*, v. 21, 277.
MONTEL, R. & COUPUT, A. (1932) *Bull. Soc. Path. Exot.*, v. 25, 1032.
MONTEL, M. L. R., MASSARI, P. & LE-VAN-PHUNG. (1934) *Bull. Soc. Méd.-Chirurg. Indochine*, v. 12, 477. [*Trop. Dis. Bull.* 1935, v. 32, 55.]
MOSS, W. L. & BIGELOW, G. H. (1922) *Bull. Johns Hopkins Hosp.*, v. 33, 43. [*Trop. Dis. Bull.* 1923, v. 20, 172.]

PLEHN, A. & MENSE, K. (1924) Handbuch der Tropenkrankheiten, 3rd edit., v. 2, 622. Leipzig: Johann Ambrosius Barth. [*Trop. Dis. Bull.* 1925, v. 22, 84.]
POLAK, H. J. (1927) *Arch. f. Schiffs- u. Trop.-Hyg.*, v. 31, 530. [*Trop. Dis. Bull.* 1928, v. 25, 657.]
POWELL, A. (1923) *Proc. Roy. Soc. Med.* (Sect. Trop. Dis. & Parasit.), v. 16, 15. [*Trop. Dis. Bull.* 1923, v. 20, 835.]
RAMSAY, G. C. (1925) *J. Trop. Med. & Hyg.*, v. 28, 85. [*Trop. Dis. Bull.* 1925, v. 22, 546.]
RAT, J. N. (1891) Yaws: its Nature and Treatment. p. 12. London: Waterlow & Sons.
SCHÖBL, O. (1928) *Philippine J. Sci.*, v. 35, 209. [*Trop. Dis. Bull.* 1929, v. 26, 172.]
SCHÜFFNER, W. (1907) *Münch. med. Woch.*, v. 44, 1366.
SERRA, G. (1937–8) *Chir. organi. Mov.*, v. 23, 219.
SIMPSON, T. (1938) *West African Med. J.*, v. 10, 14. [*Trop. Dis. Bull.* 1940, v. 37, 21.]
SOETOMO, R. & EICHHORN. (1925) *Geneesk. Tijdschr. v. Nederl.-Indië*, v. 65, 207. [*Trop. Dis. Bull.* 1926, v. 23, 18.]
D'SOUZA, A. J. (1933) *Indian Med. Gaz.*, v. 68, 155. [*Trop. Dis. Bull.* 1933, v. 30, 595.]
SPITTEL, R. L. (1923) Framboesia Tropica (Parangi of Ceylon). pp. 22, 26. London: Baillière, Tindall & Cox. [*Trop. Dis. Bull.* 1923, v. 20, 263.]
STRONG, R. P. & SHATTUCK, G. C. (1930) The African Republic of Liberia, v. 1, 277. Cambridge, U.S.A.: Harvard University Press.
——. (1944) Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases. p. 392. London: H. K. Lewis & Co., Ltd. [*Trop. Dis. Bull.* 1945, v. 42, 421.]
TAKASAKI, S. (1932) *Jahrb. f. Kinderheilk.*, v. 134, 169. [*Trop. Dis. Bull.* 1932, v. 29, 722.]
TODD, K. W. (1931) *Trans. Roy. Soc. Trop. Med. & Hyg.*, v. 25, 65. [*Trop. Dis. Bull.* 1931, v. 28, 929.]
VAN NITSEN, R. (1920) *Ann. Soc. Belge de Méd. Trop.*, v. 1, 39. [*Trop. Dis. Bull.* 1921, v. 18, 251.]
VISWALINGAM, A. (1922) *Indian Med. Gaz.*, v. 57, 172. [*Trop. Dis. Bull.* 1923, v. 20, 182.]
WILLIAMS, E. R. (1939) In Shanks, Kerley and Twining's A Textbook of X-ray Diagnosis, v. 3, 450. London: H. K. Lewis & Co., Ltd.
WILSON, P. W. (1924) *U.S. Nav. Med. Bull.*, v. 20, 190. [*Trop. Dis. Bull.* 1924, v. 21, 366.]
—— & MATHIS, M. S. (1930) *J. Amer. Med. Ass.*, v. 94, 1289. [*Trop. Dis. Bull.* 1930, v. 27, 708.]
WOLTER, A. (1934) *Klin. Woch.*, v. 13, 846. [*Trop. Dis. Bull.* 1934, v. 31, 825.]

MALARIA.

CALDWELL, J. D. **Malaria Control in the Dominican Republic.** *Puerto Rico J. Pub. Health & Trop. Med.* 1945, Dec., v. 21, No. 2, 193–200. [Spanish version 201–8.]

The Dominican Republic (Santo Domingo) occupies the eastern two-thirds of the island of "Hispaniola" (Haiti) lying to the east of Cuba and Jamaica in the West Indies. The climate is typical of the Caribbean region, with a rainy season between May and October, heaviest in the spring and autumn and totalling 100 inches per annum in some parts. Malaria is prevalent, varying in intensity in different communities; spleen rates vary from 0 to 42·8 per cent. Among 1,374 positive blood smears 72·8 per cent. showed *Plasmodium falciparum*; 17·18 per cent. *Plasmodium vivax*; 4·73 per cent. *Plasmodium malariae*; and 5·61 per cent. mixed strains. *Anopheles albimanus* is the principal malaria vector. Streams are the main breeding places, but swamps, lagoons and irrigation systems may also be involved. A Malaria Division of the Health Service was formed in 1941. In relation to certain urban areas, where seasonal clinical malaria occurs, permanent major and minor drainage schemes have been instituted. The author states that requisite personnel are being trained and that larvicidal control is to be commenced.

R. Ford Tredre.

BERBERIAN, D. A. **The Species of Anopheline Mosquitoes found in Syria and Lebanon. Their Habits, Distribution and Eradication.** *J. Palestine Arab Med. Ass.* 1946, July, v. 1, No. 5, 120–46, 1 map & 10 figs. [Numerous refs.]

To enable the reader to understand the problem of anophelism in Lebanon and Syria the author outlines the main topographical and climatic features of the territory. He then gives an account of the morphology, distribution and habits of the anopheline mosquitoes, comments on their importance as vectors of malaria, and discusses the possibility of their eradication from the country.

Two mountain ranges run parallel to the sea coast ; the western range comprises the Lebanon mountains and the eastern, the Anti-Lebanon range. To the west of the Lebanons is the coastal belt ; between the ranges is the highly cultivated Bukâ plain, and east of the Anti–Lebanons is the Syrian desert. The Euphrates river passes through the north-eastern parts, the Orontes rises in the Bukâ and flows north, the Litany also rises in the Bukâ but flows south and the Barada flows east through Damascus. Many rivers cross the coastal strip ; the majority are small, stony streams, which dry up in late summer. Coastal swamps exist at the river-mouths and there are a number of inland swamps and lakes. Ancient and modern irrigation canals, cisterns, wells, holes and man-made depressions are scattered all over the countryside.

The climate of the coastal belt is generally sub-tropical, that of the mountains temperate, and that of the Bukâ extreme. From May to October heat and dryness increase from west to east ; and little moisture crosses the Anti-Lebanons which form a barrier between Syria and the Syrian desert. Wind is generally from the west and south-west.

The species of *Anopheles* are *A. sacharovi*, *A. superpictus*, *A. claviger*, *A. sergenti*, *A. hyrcanus*, *A. algeriensis*, *A. marteri* and *A. multicolor*. The first seven are established, but *A. multicolor*, though common in Palestine, has only been taken once north of the border. The first three are important vectors of malaria. Keys are provided for the identification of adults and larvae and a table is given of characters for the separation of the closely allied *A. claviger*, *A. algeriensis* and *A. marteri*. The figures are unfortunately rather indistinctly reproduced.

A. sacharovi is capable of maintaining intense malaria ; it breeds in inland and coastal swamps and in springs, cisterns and pools where the water surface is covered with a layer of vegetation. In winter, females settle in houses, barns and stables near a food supply, from whence they emerge on warm days to feed and then retire to their hiding places. *A. sacharovi* is a strong flier and in the Bukâ may travel five to ten kilometres aided by the wind.

A. superpictus transmits much of the rural malaria, particularly in the coastal belt. Larvae are commonest from May to October in the sunlit parts of mountain streams, among stones and boulders ; in springs, ditches and irrigation channels ; they also occur in water which collects in sand and gravel pits. *A. superpictus* breeding-places are flooded out in winter, consequently control measures need to be repeated each season. Adults enter houses and stables at night but their flight range is limited, possibly to a maximum of two kilometres.

In towns and villages *A. claviger* is the important species and breeds in wells, though if the spleen rate is over 10 per cent. breeding places of other species may be implicated. Introduction of piped water supplies has sometimes worsened the situation because the disused wells have remained untreated. In rural areas shaded parts of rivers and mountain streams also breed *A. claviger*, which is the first anopheline to appear in springtime.

The transmission of malaria in this region by *A. sergenti* is not confirmed, but it must be regarded as a potentially dangerous species. Larvae are sometimes found with those of *A. superpictus* in stream beds, canals and ditches, but not in marshes. Its flight range is not great and it is thought to overwinter as an adult.

The remaining species are unimportant; *A. hyrcanus* and *A. algeriensis* breed in marshes and will bite in sunlight. *A. marteri* breeds in shaded pools in hilly country; *A. multicolor* has not established itself in Syria and Lebanon.

The author suggests that the attack on the three malaria vectors should consist in spraying winter quarters of *A. sacharovi* and *A. superpictus* with 5 per cent. DDT in kerosene or 5 per cent. DDT emulsion, once in December or January and once in March, followed by treatment (with paris green, malariol or DDT) of all potential breeding places once a fortnight. In the case of *A. claviger* he suggests that wells and cisterns be sprayed every two months; in rural areas he considers that the *superpictus* control measures will suffice for the *claviger* problem also. He believes that it is possible to devise and execute such an extensive programme, and proposes that advice and direction should be sought from those who dealt with the eradication of *A. gambiae* from Brazil and Egypt. *H. S. Leeson.*

KALANDADZE, L. P. & SAGATELOVA, I. S. [**Concerning Dry Hardiness of Mosquito Larvae on Dry and Humid Substrata.**] *Med. Parasit. & Parasitic Dis.* Moscow. 1945 [1946], v. 14, No. 6, 19–25. [In Russian.]

Working with larvae of *Anopheles maculipennis maculipennis*, *A. bifurcatus* [*A. claviger*] and *Culex pipiens*, the authors carried out a series of experiments with the view to determining their viability on dry and humid substrata; the striking term dry-hardiness is used by the authors. Batches of 20 larvae of each age-group were placed in 100 cc. crystallizers containing, at the bottom, a layer of earth, sand, plants, chips of wood or a filter. After exposure for different periods of time, water was added to the substratum, and on the following day a count was made of the percentage of live and dead larvae. In a control crystallizer water was added at the beginning of the experiment. Experiments on humid substrata were carried out with a special apparatus [see this *Bulletin*, 1944, v. 41, 916.]

The results were as follows:—(1) The dry-hardiness of mosquito larvae is considerably less that that of the eggs; (2) the dry-hardiness of larvae of stages I and II is less than that of larvae of stages III and IV; (3) larvae of *Culex* are more dry-hardy than those of *Anopheles*; (4) at lower temperatures of the air the hardiness of larvae increases both on dry and humid substrata; (5) the dry-hardiness of larvae on humid substrata (33·3 per cent. moisture in the soil) is considerably higher than that on perfectly dry substrata. Thus the larvae of *A. bifurcatus* of stages II, III and IV can live from 3 to 7 days on humid substrata at 23–24°C. and 59–75 per cent. relative humidity, whereas on dry soil they survive only up to 35 minutes at 24·2°C. *C. A. Hoare.*

FEDERATED MALAY STATES: INSTITUTE FOR MEDICAL RESEARCH. **The Anopheles of Malaya. Keys for the Identification of the Females and Larvae of the Anopheles Mosquitoes found in the Malay Peninsula.** 48 pp., numerous illustrations. 1945. [Prepared by the Staff of the Entomological Division of the Institute for Medical Research, F.M.S.]

This handy little booklet is a practical introduction to the identification of Malayan anophelines; it will be useful both to medical and non-medical investigators.

The text is simple and accurate. Detailed descriptions of mosquito anatomy are not included but the important morphological features are clearly indicated in the diagrams. In thirty-seven pages the reader is shown how to recognize mosquitoes, how to distinguish culicines from anophelines and the different species of *Anopheles* from one another. Instructions are given for using the keys for the identification of females and larvae of Malayan species. The keys are followed by brief descriptions of eighteen important species, notes on their habits, distribution, breeding places and relation to malaria, and a brief glossary of some technical terms.

In the few remaining pages there is a short account of malaria in the Malay peninsula and a description of the man-baited double-net method of trapping mosquitoes. *H. S. Leeson.*

DAGGY, R. H. **The Biology and Seasonal Cycle of** *Anopheles farauti* **on Espiritu Santo, New Hebrides.** *Ann. Ent. Soc. Amer.* 1945, v. 38, No. 1, 1–13. [Summary taken from *Rev. Applied Entom.* Ser. B. 1946, Aug., v. 34, Pt. 8, 122–3.]

Anopheles farauti, Lav.,* the mosquito formerly known as *A. punctulatus moluccensis*, Sw. & Sw.,* occurs in the New Hebrides (including the Banks Islands), the Solomons, Bismarck Archipelago, Admiralty Islands and eastern New Guinea and is the most important vector of malaria in the Australasian region. It was studied from October 1942 to September 1943 in Espiritu Santo (New Hebrides), where it is the only Anopheline and malaria is hyperendemic. An account is given of the precipitation, geological structure and drainage of the island. *A. farauti* is primarily a coastal mosquito, though the author found it to be abundant at an altitude of 1,100 ft. on Gaua Island in the Banks Group. Permanent breeding places in Espiritu Santo include rivers, streams, springs, taro gardens, seepage areas, ponds, lakes, swamps, marshes, open wells and fresh-water and slightly brackish lagoons. During the rainy season, breeding occurs also in such temporary sites as pools, ruts and hoofprints. The breeding places vary widely in respect to sunlight, temperature, plant growth, turbidity and movement. Breeding and malaria transmission occur throughout the year wherever water is available, but in the south-eastern part of the Island, where there are few permanent breeding places, transmission takes place principally in the rainy season. At this time, adults are produced in large numbers in all breeding places except some of the larger rivers, from which the larvae are flushed by frequent floods. It is estimated that troops were responsible, principally through the construction of roads and the movement of heavy equipment, for the formation of 90 per cent. of the temporary breeding places in the area in the south-east that has been occupied by them. Moreover, the suitability of some permanent water for breeding was increased. The natives favour the transmission of malaria by building villages near rivers and by making water terraces for growing water taro, a staple food crop.

The flooding of low-lying fresh-water marshes with sea water was considered as a possible control measure, but investigation showed that the maximum salinity at which breeding can occur is high. Females given a choice of 11 dishes containing water of different salinities varying from rain water to pure sea water oviposited in almost all, including the pure sea water, though they seemed to have some preference for concentrations of less than 50 per cent. sea water. Eggs hatched in pure sea water, but the larvae died in a few hours.

* It appears from a recent key that American authorities have concluded that *A. farauti* is specifically distinct from *A. punctulatus*, Dön., and that *moluccensis*, if, as is probable, it is a distinct form, is a sub-species of *farauti* and not of *punctulatus*.

However, complete development took place in water containing 13,000 parts chlorides per million (65 per cent. sea water), larvae were found in nature in water containing 13,500 parts chlorides per million (68 per cent. sea water) and larvae taken from such water in the second or third instar completed their development in partly evaporated sea water containing 23,000 parts chlorides per million. Opening low-lying areas to sea water may therefore increase the area available for breeding, but where tidal flushing is marked and the water is not impounded, larvae are greatly reduced in numbers or eliminated.

In individuals reared in the laboratory at 28–30°C. [82·4–86°F.] on dog biscuit, the egg, larval and pupal stages lasted 1½–2, 10 and 1½ days, respectively. The cycle in open, sunlit pools is probably shorter. The females attack man readily, though they also feed on animals. In houses or tents with artificial light, they bit chiefly in shaded corners or on shaded parts of the body. They fed principally, but not exclusively, at night. Their bite is painless and their flight relatively quiet. They sometimes used artificial shelters, such as dwellings, tents, boxes and large tins, as diurnal resting places, but were also found on the walls of slit trenches and the underside of logs. They were never found, however, in standard boxes placed in favourable situations in attempts to estimate adult populations. From observations on the distances between areas in which malaria occurred in the dry season and the nearest breeding places, it was estimated that the effective flight-range of the females is ¾–1 mile, and intensive control measures within this radius considerably reduced the malaria rate. A female was found, together with mosquitos of other species, on a boat 600 yards offshore where the crews of several boats were reporting mosquito annoyance at night and becoming infected with malaria.

Filariasis with nocturnal periodicity is endemic in the New Hebrides. Of the mosquitos occurring there, the only ones in which the complete development of *Filaria* (*Wuchereria*) *bancrofti* has been observed are *A. farauti* and *Culex fatigans*, Wied. (*quinquefasciatus*, auct.), and as the latter is recently introduced and rare, *A. farauti* is probably the important vector.

EADS, R. B. **A New Record of *Anopheles albimanus* in Texas.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 420.

"Five *Anopheles albimanus* females were taken within a two-week period from three different locations near the Corpus Christi Naval Air Station. This represents the first known migration of this species from the Lower Rio Grande Valley into other parts of Texas."

PACKER, H. **The Use of Darkfield Illumination in Studies of Malaria Parasites.** *J. National Malaria Soc.* Tallahassee, Fla. 1945, Dec., v. 4, No. 4, 331–40, 5 figs.

In this paper the author gives an account of his experience with the use of dark field illumination for the study of malaria parasites. He describes the apparatus he has found most useful and his method of preparing blood for examination. He advocates the admixture with the blood of a little distilled water or tap water by inverting a small drop of water on the coverglass on to the drop of blood on the slide. Rapid detection of *Plasmodium vivax* was possible owing to the brilliant refractiveness of its pigment which was in active Brownian movement. The method is less satisfactory for *P. falciparum* owing to the paucity of pigment in the young rings which are the forms in the peripheral blood. Observations are readily made on the process of exflagellation. The author concludes that when blood examinations are intended for the discovery of *P. vivax* alone the dark field method is often the most rapid for obtaining a diagnosis. *C. M. Wenyon.*

GUHA, G. S. **A Case of Quinine Amblyopia.** *Indian Med. Gaz.* 1946, June–July, v. 81, Nos. 6/7, 238–41, 5 charts.

HALAWANI, A. & NOR-EL-DIN, G. **A Case of Raynaud's Disease-like Condition of Unusual Severity following Intramuscular Quinine Injections.** *J. Roy. Egyptian Med. Ass.* 1946, Jan.–Feb., v. 29, Nos. 1/2, 84–9, 2 figs.

"1. A case presenting the signs and symptoms of Raynaud's syndrome was described. The patient was a female Mohamedan aged fifteen years from the Dakhla Oasis of the Egyptian Western Desert. She was suffering from chronic malaria and syphilis, but there was no haemoglobinuria.

"2. The points of interest of this case are the close relation between the administration of quinine in the gluteal muscles and the onset of the lesion, the severity of the condition (leading to gangrene with complete loss of the toes), and its occurrence in only one limb and that limb being the lower extremity."

LANGE, K. & MATZNER, M. J. **The Distribution of Atabrine in the Blood, the Skin, and its Appendages. Methods for the Rapid and Simple Detection of the Presence of Atabrine in Blood, Skin, and Nails.** *J. Lab. & Clin. Med.* 1946, July, v. 31, No. 7, 742–8, 4 figs. [10 refs.]

The authors have used a single extraction method in conjunction with a fluorocomparator to estimate plasma levels of atebrin. The earlier methods described for the estimation of this substance [this *Bulletin*, 1936, v. 33, 273; 1938, v. 35, 103, 569] lacked sensitivity, while the methods of BRODIE and UDENFRIEND and of MASEN [this *Bulletin* 1944, v. 41, 453; 1945, v. 42, 8] which meet all requirements, demand considerable skill and laboratory resources. The present method consisted in basifying the plasma and extracting atebrin base with ether. Fluorescence in the extract was then measured by a fluorophotometer or visual fluorocomparator with selective light filters to cut off undesired fluorescence; repeated extraction with solvents was thus avoided. The technical details of the instruments are described. By the above means the plasma levels in 22 patients receiving atebrin for treatment of relapsing *P. vivax* infections were studied. The authors found that 0·3–0·6 gm. atebrin on the first day followed by 0·2 gm. daily for 7 days was not sufficient to give a desired plasma level of 100 microgrammes per litre, but that this level can be reached in 24 hours if 1·2 gm. is given on the first day followed by 0·6 gm. on the following day and 0·3 gm. daily thereafter during treatment. Use was also made of ultra-violet light of wave-length 3,600 Ångstrom units to excite fluorescence in the skin and appendages of atebrin-treated patients which could be observed visually or measured quantitatively by a photo-electric Dermofluorometer previously described. No direct correlation was found between the amounts of atebrin present in plasma and skin. Observation of the fluorescence in nails readily indicated whether atebrin was being taken regularly. *J. D. Fulton.*

SMITH, L. H. & STOECKLE, J. D. **Effect of Quinacrine Hydrochloride (Atabrine) on Isolated Mammalian Heart.** *Proc. Soc. Exper. Biol. & Med.* 1946, June, v. 62, No. 2, 179–84, 2 figs. [13 refs.]

A study was made of the effect of adding mepacrine to the heart-lung preparation of dogs. Contractility of the heart muscle was impaired, so that the output of the heart diminished and the volume of the heart increased. The outflow from the coronary sinus did not change significantly. Bradycardia occurred sometimes, but not always. The electrocardiograph was approximately normal. These changes could be reversed by adding cardiac glucosides

(ouabain or K-strophosid). The minimal toxic concentration of mepacrine was about 1 mgm. per litre of whole blood ; 1·5 mgm. per litre was regularly toxic. Much of the mepacrine was rapidly removed from the circulating blood ; 50 per cent. disappeared from the blood in 15 minutes in one experiment ; in another, 95 per cent. disappeared in about 50 minutes, although the heart and lungs are the only tissues available in the preparation to take it up. Rapid injection of mepacrine leads to severe heart failure, but a slow infusion of the same amount can readily be tolerated. Accordingly, if mepacrine is to be given intravenously it should be given extremely slowly. *F. Hawking.*

GUTIÉRREZ SÁNCHEZ, Elena. Acumulación y excreción de la atebrina en el ratón. [**Concentration and Excretion of Atebrin in Mice.**] *Rev. Inst. Salubridad y Enfermedades Trop.* Mexico. 1946, Mar, v. 7, No. 1, 1–12, 1 graph [14 refs.] [Summary in English by the author.]

" A study was made of the concentration of atabrine chlorate [*sic* : in the original it is Clorhidrato] in the blood of white mice : its excretion in the faeces and urine, and its storage in the liver, spleen, lungs, heart, intestine and kidneys. The drug was administered orally 113 mgs. per kilogram of weight, 24 mgs. intravenously and 25 mgs. intraperitoneally, in a single dose and repeated doses.

" The atabrine concentration in the blood with one daily dose for several days, never exceeded 0·50 mgs. per cent., but this low concentration continued for five days after treatment was suspended.

" High concentrations of the drug were found in the liver, disappearing completely eight days after the treatment was suspended. Lower concentrations were found in the spleen and lungs."

BASFORD, F. R., CURD, F. H. S. & ROSE, F. L. **Synthetic Antimalarials. Part VIII. Some 4-Arylamino-6-Aminoalkylamino-2-Methylpyrimidines.** *J. Chem. Soc.* 1946, Aug., 713–20.

CURD, F. H. S., DAVIS, M. I., OWEN, E. C., ROSE, F. L. & TUEY, G. A. P. **Synthetic Antimalarials. Part IX. 4-Arylamino-2-Aminoalkylamino-6-Methylpyrimidines. Further Variations.** *J. Chem. Soc.* 1946, Aug., 720–29.

CURD, F. H. S. & ROSE, F. L. **Synthetic Antimalarials. Part X. Some Aryl-Diguanide (" -Biguanide ") Derivatives.** *J. Chem. Soc.* 1946, Aug., 729–37.

KAPLAN, L. I., READ, H. S. & BECKER, F. T. **The Action of Thiobismol on Therapeutic Quartan Malaria.** *J. Lab. & Clin. Med.* 1946, July, v. 31, No. 7, 735–41, 1 fig.

It was shown by SCHWARTZ (*J. Pharm. & Exper. Therap.* 1939, v. 65, 175) that sodium bismuth thioglycollate (thiobismol Bi $(SCH_2COONa)_3$ had some effect in controlling the number of paroxysms during therapeutic malaria treatment with *P. vivax.* YOUNG *et al.* [this *Bulletin*, 1943, v. 40, 881] failed to obtain consistent results with *P. malariae* and the drug was apparently without action on *P. falciparum.* Investigation has now been made with this drug in 38 patients with blood-induced *P. malariae* infections who were experiencing true quartan, double quartan or quotidian attacks, in order to find out at what stage of growth the parasites are affected and also its effect on the paroxysms. Fifty-six injections of the drug in 0·1 or 0·2 gm. doses were given intramuscularly at irregular intervals. Its controlling effect on the paroxysms was decided by relating the time of administration to the growth stage of the parasite in all types of cycle encountered. Young or mature forms of the parasite were

practically unaffected. In about half the patients with true quartan cycles the drug exerted a definite clinical effect because of its action on immature forms of the parasite. Paroxysms were interrupted in a very large percentage of patients experiencing the other types of cycle and the partially mature forms of the parasite were chiefly affected. Patients with parasite counts above 10,000 per cmm. showed no clinical response to treatment, but the parasite numbers were reduced. The authors conclude that in quartan malaria the drug is of value in reducing the frequency of paroxysms and in controlling irregular cycles. It should aid neurosyphilitics undergoing treatment to tolerate better a full course of therapy with this parasite. *J. D. Fulton.*

CLUVER, F. W. P. **Report on Malaria Control in Rural Native Areas by using D.D.T. Insecticide.** *South African Med. J.* 1946, July 13, v. 20, No. 13, 368–70.

The author, working in a Zululand Reserve, reports on the substitution of weekly insecticidal spraying of native huts with Pyagra paraffin by the alternative method of 5 per cent. DDT in commercial paraffin. The local anopheline vectors of malaria are *Anopheles gambiae* and *A. funestus*. The results in an admittedly small-scale experiment illustrate the inefficiency of a weekly pyrethrum spraying programme when compared with the degree of adult mosquito eradication attained with DDT.

Park Ross instituted insecticidal spraying in Zululand [this *Bulletin*, 1937, v. 34, 58] with resultant control of seasonal epidemic malaria ; it remains to be seen what effect the general use of residual DDT, as described by the author, will have on endemic malaria.

The author quotes a saving of 12 per cent. in costs in the replacement of weekly Pyagra by three-monthly residual DDT. The additional benefit of a heavy fly and cockroach mortality is noted. *R. Ford Tredre.*

JOHNSON, H. A. & EASON, J. L., Jr. **DDT in Paradichlorobenzene as a Larvicide.** *Pub. Health Rep.* Wash. 1946, Aug. 9, v. 61, No. 32, 1185–8, 1 fig.

DDT pellets were prepared by melting paradichlorobenzene and adding "solvents containing DDT and emulsifiers and DDT in crystalline form". Details of two of the seven formulae are given here, as examples :—

475 cc. paradichlorbenzene.
25 cc. of a 50% solution of DDT in Velsicol 70 plus 1·5% Triton X-100.
20 gm. DDT.

475 cc. paradichlorbenzene.
25 cc. of a 7% solution of DDT in No. 10 motor oil.
20 gm. DDT.

Suspension of the pellets in water in field experiments resulted in slow disintegration, 50 per cent. of volume in 14 weeks. A "marked reduction" in mosquito larvae occurred for the first six weeks, after which scum and vegetation began to interfere with the action of the DDT. The authors consider the results attained were promising and that a higher rate of pellet disintegration would result in a higher degree of *Anopheles* control. *R. Ford Tredre.*

KRUSE, C. W. & METCALF, R. L. **An Analysis of the Design and Performance of Airplane Exhaust Generators for the Production of DDT Aerosols for the Control of** *Anopheles quadrimaculatus*. *Pub. Health Rep.* Wash. 1946, Aug. 9, v. 61, No. 32, 1171–84, 5 figs.

The authors describe a method of control from the air by DDT larvicide of the larvae of *A. quadrimaculatus* under conditions encountered on the impounded

waters of the Tennessee Valley Authority. Spraying of such areas is attained by the atomization of a DDT solution by the exhust gases of the airplane engine. The larvicide is injected into a " venturi " extension of the engine-exhaust by a wind-driven pumping unit. The following desirable factors in a DDT solvent, for this use, are quoted :—

(1) High solvent power to permit the use of concentrated solutions which greatly increase " payload " ;

(2) low volatility, which increases safety factors ; this is desirable for the persistence of the solution on the water surface, and decreases evaporation of solvent due to the hot exhaust gases ; and

(3) non-corrosive and non-poisonous chemical properties.

The methylated naphthalenes were chosen, and in particular a fraction Velsicol NR-70 (Velsicol Corporation, Chicago) was readily available ; a 20 per cent. solution of DDT in the latter solvent was used.

The optimum size of aerosol droplet deposited on water surface lay between 25 and 50 microns in mass median diameter ; this particle size (35 microns) enabled 200-foot swath widths, from a height of 30 feet, to receive the adequate larvicidal dose of DDT ; a particle size of under 50 microns ensures adequate penetration of heavy vegetation cover. A deposit of 0·0001 lb. DDT per acre (1 microgramme of DDT per square foot) with a 35-micron particle size killed 90 per cent. of *A. quadrimaculatus* larvae in the open. In practical application the authors recommend the following deposit figures per acre to ensure a 90 per cent. *A. quadrimaculatus* larval kill :—Open, 0·0005 lb. DDT ; medium cover, 0·0025 lb. DDT ; high cover, 0·005 lb. DDT. A table is given showing the widths of 90 per cent. larval kill which will be obtained under varying rates of plant cover, aerosol discharge and flight pattern.

[This paper is a valuable contribution on this method of anopheline control.]

R. Ford Tredre.

BEKLEMISHEV, V. N., SHLENOVA, M. F. & ORLOV, P. M. [**Experiment on the Determination of the Necessary and Sufficient Radius of Antilarval Treatment.**] *Med. Parasit. & Parasitic Dis.* Moscow. 1945 [1946], v. 14, No. 6, 3–8, 1 fig. [In Russian.]

A knowledge of the size of the area to be covered in undertaking anti-larval measures for the protection of a given site against malaria is of great practical importance. If the radius is too small, mosquitoes continue to enter the protected zone ; if it is excessive, considerable unproductive expense is involved. In general, the area of the protected zone is proportional to the square of its radius. The actual practice varies according to local conditions. In the case of *Anopheles maculipennis* a width of 3 km. is considered to be sufficient, but in the tropics it is usually reduced to 1·6 km., while in Russia it is sometimes extended to 5 km.

Working in the flood area of the Volga, near Kuibyshev, in 1940 and 1941, the authors carried out experimental field observations on this question. The problem was to determine the effect of the width of a controlled zone surrounding a settlement, which it was desired to protect, upon the influx of mosquitoes into the settlement ; in other words to ascertain the rôle of each concentric belt around the settlement upon the mosquito density in it. With this end in view, an estimate was made of the density of larvae in an area with a radius of 3 km. from the settlement, after which complete anti-larval measures were undertaken in all the breeding places within three concentric zones, each 1 km. wide, in succession. Originally, the zones with a radius of 1 and 2 km. were found to have a uniform density of larval population, while in the outer zone (radius 3 km.) the density was much lower. After the first kilometre zone

had been dealt with, the larval density within the second kilometre zone was doubled, whereas in the third kilometre zone it increased only after treatment of the second zone. This centrifugal increase was probably due to dispersion of female mosquitoes from the water-collections treated with petroleum. While the increased density of the second zone, following treatment of the first zone, almost compensated for the depopulation of the latter, the density in the third zone was unable to keep pace with the losses caused by anti-larval treatment of the first two zones.

The results of these operations were correlated with an estimate of the density of adult mosquitoes within the settlement, based on counts made in three control sites. After treatment of the first zone, the mosquito density in the settlement was reduced by 82·4 per cent. After treatment of the second zone it fell to 0·3–0·4 per cent. of the original density. Thus, thorough larval destruction within a radius of 2 km. almost completely stopped the influx of active females (*A. maculipennis messeae*) into the protected settlement. From this experiment it would seem that the clearance of a zone within 2 km. from the protected area was sufficient. However, it is pointed out that in this instance the mosquitoes might have been deflected by two other villages within the area of operations and by cattle in the vicinity. It is concluded that, whereas in individual cases anti-larval measures might be limited to a radius of 2 km., in other cases it is advisable to cover 3 km. *C. A. Hoare.*

KNISELY, M. H., STRATMAN-THOMAS, W. K., ELIOT, T. S. & BLOCH, E. H. **Knowlesi Malaria in Monkeys. I. Microscopic Pathological Circulatory Physiology of Rhesus Monkeys during Acute** *Plasmodium knowlesi* **Malaria. A Motion Picture.** *J. National Malaria Soc.* Tallahassee, Fla. 1945, Dec., v. 4, No. 4, 285–300. [Numerous refs.]

In previous papers [this *Bulletin*, 1943, v. 40, 584] the authors have described the results of investigations, carried out by a special technique, on the behaviour of blood and the blood vessels in living *Macacus rhesus*. The observations were made on normal monkeys and on monkeys suffering from *P. knowlesi* infection. The results throw light not only on the pathology of malaria but also on the normal physiology of the blood and blood cells. To render these findings available to students and others interested in the subject the authors have prepared a film. It is a 16-millimetre silent Kodachrome picture about 1,500 feet long, on four reels, and takes from sixty to eighty minutes to project. It was taken through the microscope at magnifications of 32, 48 or 96 diameters. It shows the normal physiology of the blood and the changes which occur when there is a malarial infection leading to the formation of clumps of infected cells, which not only impede the circulation but also lead to a condition of anoxia. Finally the effect of atebrin in disintegrating the clumps is shown. The paper describes the film in considerable detail and points out that it is available on loan to medical schools, medical societies, medical officers of the military services and research groups. Applications should be made to Dr. M. H. KNISELY, Department of Anatomy, University of Chicago, or to Dr. T. S. ELIOT, Department of Anatomy, University of Tennessee, Memphis.

C. M. Wenyon.

EXEMPLARSKAYA, E. V. [**Therapeutic Effect of the Cytotoxic Antireticular Serum in Malaria of Monkeys** (*Macacus rhesus*).] *Med. Parasit. & Parasitic Dis.* Moscow. 1945 [1946], v. 14, No. 6, 34–7. [17 refs.] [In Russian.]

In recent years the Russian physiologist BOGOMOLETZ has developed a method of stimulating the defence phagocytic functions of the reticulo-endothelial system with the help of a so-called "cytotoxic antireticular serum",

which is produced by immunizing an animal with emulsion of RE tissue of another [see *Bulletin of Hygiene,* 1944, v. 19, 823].

In view of the predominant part played by phagocytosis in the defence mechanism of the host in malaria the author tested the therapeutic effect of cytotoxic serum in monkey malaria. The experiments were carried out in *Macacus rhesus* [*Macaca mulatta*] infected with *Plasmodium inui*, while the cytotoxic serum was produced in rabbits immunized with an emulsion of spleen and brain from monkeys (homologous antigen) and from dogs (heterologous antigen). The titre of the cytotoxic serum of rabbits was determined by the complement fixation test, and the serum was considered to be suitable at a titre not lower than 1 : 100. It was injected into the experimental monkeys intravenously in doses of 0·05, 0·1 and 0·2 cc. before, simultaneously with, and after induction of infection. Control monkeys either received normal heterologous serum or were infected without any serum. The therapeutic effect of the cytotoxic serum was determined on the basis of the following indications: (1) intensity of parasitaemia and severity of clinical symptoms; (2) temperature reaction; (3) stimulation of activity of reticulo-endothelial cells; (4) haematological picture; and (5) relapses.

The parasite rate was based on a count per 1,000 red cells. The most severe form of malaria, both clinically and parasitologically (6 per cent. infected red cells), occurred in monkeys which received the maximum dose of serum (0·2 cc.). It was milder in those which received 0·1 cc. of homologous serum (parasite rate 2·8 per cent.), while those injected with the minimum dose (0·05 cc.) had a very mild abortive infection with a parasite rate below 1·5 per cent. The heterologous cytotoxic serum had a relatively slight effect upon the reduction of parasitaemia. Among the control monkeys, in those given 0·05 cc. of heterologous serum the course of the disease was as severe as in those without serum, while only a slight reduction of parasitaemia occurred in those which had 0·1 and 0·2 cc. serum.

The blood picture as well as the clinical symptoms corresponded to the severity of the disease. The effect of the serum treatment upon relapses was similar to that upon the parasite rate, *i.e.* the higher the dose the greater the liability to relapse, no relapses having been observed in monkeys treated with the minimum dose of serum (0·05 cc.).

It is concluded that treatment with cytotoxic antireticular serum leads to a stimulation of the defence phagocytic function of the reticulo-endothelial system in infected monkeys, which results in a suppression of all manifestations of the infection, the maximum effect being produced by the lowest doses of the serum. *C. A. Hoare.*

HAAS, V. H., WILCOX, Aimee & EWING, Frances M. **Infection of Chick Embryos with Non-Pigmented Forms of** *Plasmodium gallinaceum.* *J. National Malaria Soc.* Tallahassee, Fla. 1945, Dec., v. 4, No. 4, 279–84.

The authors show that inoculation of an emulsion of brain substance containing exoerythrocytic stages of *P. gallinaceum* into the yolk sac of developing chick embryos leads to infections which are chiefly of exoerythrocytic forms. Erythrocytic forms are either absent or few in number. It has also been shown that subinoculation with emulsions of the brains of infected embryos leads to the same type of infection. Seven serial passages have been made in this way so that it would seem that a preponderating exoerythrocytic infection could be carried on indefinitely. In a certain number of chicks which hatched and were found to be infected, the infection was of the same type. As regards the erythrocytic forms in the embryos, it was noteworthy that many of these were without pigment. It is thought that these pigmentless forms are directly

developed from merozoites of exoerythrocytic forms. As chicks inoculated with blood of embryos containing pigmentless erythrocytic forms develop infections of the ordinary type in which the erythrocytic forms contain pigment, it is assumed that in the embryos or in the chicks found infected when hatched the pigmentless forms would have acquired the power of forming pigment had the hosts lived long enough for several cycles to have occurred. *C. M. Wenyon.*

HUFF, C. G. & COULSTON, F. **The Relation of Natural and Acquired Immunity of various Avian Hosts to the Cryptozoites and Metacryptozoites of** *Plasmodium gallinaceum* **and** *Plasmodium relictum.* *J. Infect. Dis.* 1946, Mar.–Apr., v. 78, No. 2, 99–117, 3 figs. [25 refs.]

When chickens are infected with *P. gallinaceum* by blood inoculation, after an incubation period the length of which depends on the dose of infected cells administered, there develops an infection the intensity of which varies with the size of the inoculum. Exoerythrocytic forms begin to appear at about the eighth day and they reach a maximum at about the 25th day. The chickens may die at this stage, though very few or no erythrocytic parasites are present in the blood following the falling off of the blood infection at the crisis. A relapse of the blood infection, of short duration, may occur, and if the bird survives, the exoerythrocytic stages are rapidly reduced. When sporozoites are injected instead of blood, blood infections result whose intensity varies with the dose. When the dose of sporozoites is small the resulting blood infection may be difficult to detect. In any case there is an incubation or prepatent period, which varies little (5 to 8 days). Following the injection of sporozoites, pre-erythrocytic stages are very abundant in the tissues. When canaries are inoculated with blood or with sporozoites, no blood infection occurs, and exoerythrocytic stages cannot be found in the tissues. The canary is, therefore, completely immune to *P. gallinaceum* infection.

Ducks and geese occupy an intermediate position. Blood inoculation produces a transient blood infection but no exoerythrocytic stages can be found. When sporozoites were injected, geese showed a transient parasitaemia but the presence of parasites in the blood of the duck was demonstrable only by inoculation of 1-5 cc. of blood into chicks. In ducks, following blood or sporozoite inoculations, parasites might persist in the blood, as proved by chick inoculations, for as long as 8 months. No exoerythrocytic stages could be found in the tissues. Some experiments carried out with guinea fowl showed that those birds behaved in the same way as the ducks. These observations showed that ducks, geese and guinea fowl occupied an intermediate position between chickens and canaries as regards susceptibility to *P. gallinaceum.*

It was of interest to discover whether this relative immunity of these birds had any influence on the development of sporozoites. When sporozoites are injected into the skin of the wing of chickens their development into cryptozoites and metacryptozoites (pre-erythrocytic stages) can readily be followed at the site of inoculation. When this is carried out in ducks and geese the complete development takes place as in chickens. In canaries, as noted above, no such development occurs. It seems clear that the natural immunity of the duck and goose, which influences the blood infection, has no effect on the development of the pre-erythrocytic stages. Attempts were made to render three ducks more susceptible by inoculating them repeatedly with chicken's blood before injecting sporozoites. In two, parasites were seen in the blood in small numbers during three days. In some cases at least these parasites appeared to be in chicken erythrocytes. Conversely, attempts to reduce the susceptibility of chickens by inoculations of ducks' blood were not very convincing. When inoculated with sporozoites the chickens developed modified blood infections—an observation

which proved at least that invasion of the chicken erythrocytes occurred, though they were bathed in duck plasma.

In another series of experiments the development of sporozoites in hyper-immunized chickens and ducks which had recovered from primary infections was studied. In both birds the injection of sporozoites into the skin of the wing was followed by their complete development into cryptozoites and metacryptozoites, as in normal birds. It was again evident that the acquired immunity, though influencing the erythrocytic infection, had no effect on the pre-erythrocytic development. The experiments with *P. gallinaceum* in chickens, ducks and geese were repeated with *P. relictum* in canaries and pigeons. Injection of sporozoites from *Culex pipiens* into the skin was followed by complete pre-erythrocytic development, and in the canary by heavy blood infections. In the pigeon, however, there was no visible blood infection. In one pigeon which had had a latent infection for a year, sporozoites developed at the site of inoculation into the skin of the wing as they did in normal birds.

From the results of the experiments the authors conclude that both natural and acquired immunity are more effective against the erythrocytic than the pre-erythrocytic stages. The observations did not extend to the exoerythrocytic forms which occur late and which appear in the tissues after infections have been produced by blood inoculations, as with *P. gallinaceum* and *P. cathemerium*. These stages are not necessarily related to developmental stages of sporozoites. To distinguish them from the pre-erythrocytic cryptozoites and metacryptozoites which are the direct result of sporozoite development, the term phanerozoite is introduced. Though this term may be applied to all exoerythrocytic stages occurring in blood-induced infections, a distinction between metacryptozoites and panerozoites in sporozoite-induced infections is difficult to make. The exoerythrocytic stages may again be classified according to the type of cell invaded. Thus in *P. gallinaceum* the cells are of the lymphoid macrophage system, in *P. elongatum* they are the blood and blood-forming cells of the myeloid series, while in *P. mexicanum* of the lizard both groups of cells are involved. On this basis it would be possible to group exoerythrocytic stages as of the *gallinaceum* type, the *elongatum* type or the *mexicanum* type.

Another point raised is the nature of the natural or acquired immunity. Is this dependent upon one or more antibodies which influence the erythrocytic and various exoerythrocytic stages differently? The authors state that only one may be involved, but that in order to understand fully the immune reactions it will be necessary in the first place to concentrate observation on each individual stage. When sporozoites are injected there is extensive invasion of endothelial and lymphoid-macrophage cells by metacryptozoites before erythrocytic infection occurs. Between these two events there is a delay which is called the tissue-blood barrier. When the barrier has been breached, rapid progress of the blood infection occurs with development of immunity against this stage. As no cryptozoites or metacryptozoites occur in canaries inoculated with sporozoites of *P. gallinaceum*, it has to be concluded that the barrier exists in macrophages or similar cells, or in antagonistic humoral substances.

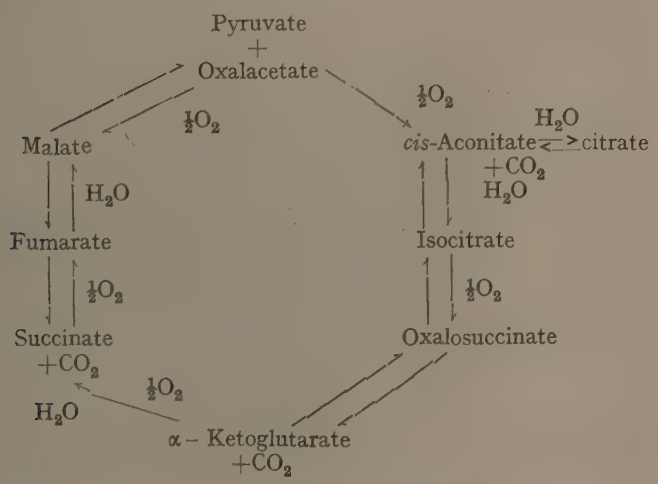
C. M. Wenyon.

SPECK, J. F., MOULDER, J. W. & EVANS, E. A., JR. **The Biochemistry of the Malaria Parasite. V. Mechanism of Pyruvate Oxidation in the Malaria Parasite.** *J. Biol. Chem.* 1946, July, v. 164, No. 1, 119–44. [57 refs.]

Observations on the metabolism of malaria parasites within red cells, or in the free state, have been reviewed [this *Bulletin*, 1938, v. 35, 709; 1940, v. 37, 190, 509; 1942, v. 39, 397, 742; 1943, v. 40, 674; 1945, v. 42, 448]. Various acid products formed from carbohydrate by these organisms have been detected.

Recent studies by Speck and Evans in this series [this *Bulletin*, 1945, v. 42, 967] showed that cell-free extracts of *P. gallinaceum* cause glycolysis by reactions similar to those occurring with yeast and muscle, in which glucose is oxidized through pyruvate as an intermediate. The mechanism of pyruvate oxidation by this plasmodium in parasitized cells and in the free state has now been studied. The authors have found that both types of parasite preparation may bring about the complete oxidation of pyruvate, and consider from experimental evidence that the reaction occurs through the well-known tricarboxylic acid cycle of KREBS, which is represented diagramatically as follows :—

Tricarboxylic Acid Cycle



Evidence was also obtained that glucose and lactate are oxidized by the same pathways as pyruvate. The parasitized red cells were obtained by cardiac puncture of chickens at the peak of parasitaemia and were separated by centrifugation, washed in calcium-free phosphate-saline at *p*H 7·4 and suspended in the same medium. The best preparations of free parasites were obtained by the use of specific haemolytic serum prepared by injecting rabbits with normal erythrocytes ; guineapig serum was used as complement. Gas exchanges during metabolism were recorded in a Barcroft-Warburg apparatus. It was noted by earlier workers that appreciable respiration by the parasites occurred in the absence of added substrate. During this phase the present authors have found that the R.Q. is about 0·9, that malonate causes inhibition of respiration and that small amounts of succinate are formed. They believe that the unknown substrate is readily replaced by others more easily oxidized. The addition of dicarboxylic acids and certain water-soluble cofactors such as thiamin, phosphate esters or mixtures of cofactors, as well as a low concentration of manganous ions and various tissue extracts increased the rate of oxygen consumption and pyruvate oxidation by the free parasites. Parasitized red cells did not require cofactors. A number of water soluble vitamins, including flavins, were without effect on pyruvate oxidation, but it is known that such substances have some metabolic function in malaria parasites. The marked increase of oxygen consumption caused by extracts of bakers' yeast was ascribed to its high content of succinic acid. On comparing the metabolic

activities of parasitized erythrocytes and free parasites it was found that the former oxidized glucose, lactate, pyruvate, succinate and malate more rapidly. The lactic acid formed by the free parasites from glucose markedly reduced oxygen consumption. Both parasite preparations showed similar glycolytic activities. Parasitized red cells were, however, unable to oxidize a number of the acids of the tricarbolylic acid cycle, possibly because of the fact that the red cell is impermeable to them. Pyruvate was oxidized by both only under aerobic conditions, and while parasitized cells effected complete oxidation with formation of only traces of acetate, free parasites produced the latter substance in considerable amounts, possibly on account of damage to enzymes involved in the above cycle, or alternatively there may be two pathways for pyruvate oxidation. In the case of *P. gallinaceum* under the above conditions, acetate appears to be an end-product of carbohydrate metabolism, but with intact cells under physiological conditions its formation may not be of importance. The formation of very small amounts of citrate (also produced by normal red cell preparations) and succinate was also detected. In contrast to the stimulation of pyruvate oxidation by some dicarboxylic acids, such oxidation was markedly inhibited by malonate accompanied by accumulation of succinate, and this gave weight to the belief that pyruvate is oxidized through the tricarboxylic acid cycle. It was noted, however, that citrate and *cis*-aconitate involved in this cycle were not utilized by the parasites. A consideration of oxygen consumption by parasitized red cells suggested that the intact erythrocyte was impermeable to some of the acids involved in the above cycle, with the result that they failed to catalyse the oxidation of pyruvate. Malonate also caused inhibition of complete oxidation of glucose and lactate as in the case of pyruvate, with formation of acetate. The oxidation pathway for these three substances is probably the same. The conversion of glucose to pyruvate by *P. gallinaceum* appears to involve phosphorylating reactions, the pyruvate formed being completely oxidized in the tricarboxylic acid cycle, or reversibly reduced to lactate. The conversion of pyruvate to acetate has also been noted as an alternative pathway of utilization.

[The experiments described in this series of papers provide important contributions to the subject of malaria parasite metabolism, and the reviewer is aware that an abstract may sometimes be inadequate to do them justice.]

J. D. Fulton.

MOULDER, J. W. & EVANS, E. A., Jr. **The Biochemistry of the Malaria Parasite. VI. Studies on the Nitrogen Metabolism of the Malaria Parasite.** *J. Biol. Chem.* 1946, July, v. 164, No. 1, 145–57, 1 fig. [16 refs.]

In extension of the authors' studies on the biochemistry of the malaria parasite, the nitrogen metabolism of *P. gallinaceum* has now been investigated. The parasitized material was obtained as described in preceding parts of this series, and by using the authors' method [this *Bulletin*, 1945, v. 42, 967] cell-free extracts of the parasite, with only traces of haemoglobin, were prepared. Gas exchanges were measured in the Barcroft-Warburg apparatus under aerobic and anaerobic conditions, and the analytical methods employed are fully described. It was found that parasitized chicken red cells produced large amounts of nitrogen when maintained under aerobic conditions in the presence of glucose, while normal erythrocytes did not produce any; production was, however, less constant than the oxygen consumption during the period of incubation. Haemoglobin and possibly nucleoprotein were believed to be the sources of amino-nitrogen produced. When glucose was absent from the medium the production of amino-nitrogen decreased and was replaced by equivalent quantities of ammonia. This fact suggests that *P. gallinaceum* can de-aminate

amino-acids. Under anaerobic conditions and also in the presence of low concentrations of quinine or atebrin, amino-nitrogen production by parasitized red cells was much reduced, owing primarily, the authors believe, to interference with oxidative processes in the parasite. Their view was tested by observing protein hydrolysis in cell-free extracts of the parasite which hydrolyse native (horse) haemoglobin more slowly than denatured globin from the same source (not an unusual property of proteinases). Similar substrates from the chicken were hydrolysed at corresponding rates. In contrast to the results obtained with parasitized red cells, the formation of amino-nitrogen by the cell-free extract was not decreased under anaerobic conditions and only by high concentrations of quinine and atebrin. These results support the view that protein hydrolysis is associated with oxidative processes in the parasite.

J. D. Fulton.

RIGDON, R. H. **Malarial Pigment. A Consideration of the Mechanism of Elimination from the Duck.** *Amer. J. Clin. Path.* 1945, Nov., v. 15, No. 11, 489–93. [13 refs.]

The author has studied the pigments which appear in the tissues of ducks during infection with *P. lophurae*. As in other types of malaria these are of two kinds, the one giving a reaction for iron when subjected to potassium ferrocyanide and hydrochloride acid or to Turnbull's technique, the other failing to give a positive reaction under this test. It is generally supposed that the latter pigment is elaborated by the malaria parasite from the haemoglobin of the red blood corpuscle. It is considered to be ferrihaemate (haematin) and is most commonly seen in the spleen, liver and bone marrow. The pigment giving the iron reaction is haemosiderin. Though it is generally assumed that this pigment is elaborated from that portion of the haemoglobin of the infected cell not utilized by the parasite in the formation of haematin, actually it is impossible to assert that this is its only source. In the study described in this paper it was noted that in ducks examined at early stages of an infection small quantities only of haemosiderin occurred in the tissues, whereas the greatest amounts were found at late stages of an infection after all parasites had disappeared from the blood and recovery from the infection had taken place. Large quantities of haematin were present at the same time. In the liver most of the two pigments were in Kupffer cells, often very closely associated, suggesting the possibility that one might have been derived from the other. Furthermore it was noted that when the test for iron was applied the colour reaction varied from greenish-blue to blue. A reasonable explanation of this variation is that it represents degrees of oxidation of the haematin, which is completed by the formation of haemosiderin. The author considers it possible that in the endothelial cells the malarial pigment is subjected to slow oxidation leading to the formation of haemosiderin which can be utilized by the host. Very little is actually known as to the method of elimination of malarial pigment from the body, and the author's observations may explain this to some extent, if not entirely.

C. M. Wenyon.

TRYPANOSOMIASIS.

WILDE, J. K. H. & FRENCH, M. H. **An Experimental Study of *Trypanosoma rhodesiense* Infection in Zebu Cattle.** *J. Comp. Path. & Therap.* 1945, July, v. 55, No. 3, 206–28, 12 charts. [18 refs.]

Six zebu oxen were used in the experiment; they had been living for several months in an area which was concluded to be free from tsetse flies, and tests made before the experiment began indicated that they had no trypanosome infection.

The Kahama strain of *Trypanosoma rhodesiense* [this *Bulletin*, 1936, v. 33, 651] in the blood of heavily infected white rats was injected subcutaneously into the six oxen and all showed trypanosomes in their blood after 7 to 13 days. The trypanosomes appeared irregularly, never on more than six days consecutively and later only on occasional days; they were polymorphic, mostly long forms, and no posterior nuclear forms were seen.

The red and white blood corpuscles showed no variations in number that could be attributed to the infection, and the haemoglobin content never fell below 62 per cent., which is not abnormal in zebu cattle during the dry season when this experiment was made. The red cell sedimentation rate showed little change.

Plasma proteins.—These were estimated periodically throughout the experiment, which lasted for about 6 months; the proteins were fractionated with sodium sulphate by HOWE's technique (*J. Biol. Chem.* 1921, v. 49, 93 & 109) and nitrogen determinations were made with the micro-Kjeldahl method. The variations in total protein, non-protein nitrogen and pseudoglobulin were small and irregular and no appreciable change occurred in the fibrinogen; the euglobulin increased rapidly, reached maximum levels between the 6th and 15th days after inoculation, and then steadily decreased but had not reached normal levels after 20½ weeks. The albumin decreased rapidly and reached the lowest levels when the euglobulin was at the maximum. The authors conclude from these findings that infection with *T. rhodesiense* "either (a) caused an increase in the dimensional proportions of the plasma protein molecules or (b) altered the conditions of the plasma so that larger proportions of its proteins were precipitated at lower electrolyte concentrations." These changes could not be compared with those in *T. brucei* infections of oxen owing to lack of data of the latter.

The blood sugar was determined by the method of HAGEDORN and JENSEN (*Ugeskr. f. Laeger*, Copenhagen, 1918, v. 80, 1117); no hypoglycaemia was observed.

Clinical symptoms were slight; soon after infection the temperature showed certain fluctuations and some of the oxen had transient malaise, but they did not lose weight and never gave the impression of being "fly-struck".

Pathology.—Six months after the oxen were inoculated, 20 cc. of the blood of each was injected on two successive days into a goat; three of the six goats (from oxen Nos. 33, 34 and 35 respectively) showed infection. Two oxen (35 and 36) were then stunned and slaughtered and examined, microscopical sections being prepared from many organs; no trypanosomes and no pathological changes attributable to the infection were found. Guineapigs were inoculated with cerebrospinal fluid of ox No. 36 [the stunning produced cerebral haemorrhage in ox No. 35] and did not become infected.

The authors conclude that *T. rhodesiense* is only mildly pathogenic to zebu cattle, but that it is reasonable to assume "that during at least the first six months of *T. rhodesiense* infection, cattle can act as true carriers of the disease and so are a source of danger to humans when moving from areas where sleeping sickness is endemic to other tsetse fly-infected areas from which

T. rhodesiense is absent." [An attempt to infect volunteers from the cattle would have been interesting; there is, of course, now no reasonable doubt that wild and domestic animals are reservoirs of *T. rhodesiense* and their importance as such in various areas needs investigation.] *J. F. Corson.*

BURTT, E. **Observations on an Antbear** (*Orycteropus afer*) **in relation to Infection with** *Trypanosoma rhodesiense.* *Trans. Roy. Soc. Trop. Med. & Hyg.* 1946, June, v. 39, No. 6, 529–32.

The author discusses his own observations on experimental infection of an antbear with *Trypanosoma rhodesiense*, and compares them with those of VANDERPLANK [see this *Bulletin*, 1941, v. 38, 632] who suggested that the lower the normal temperature of this host before infection (in his case 97°F.), the higher was the subsequent transmissibility of the trypanosomes in tsetse flies (in his case 2 per cent. had salivary gland infections) and the shorter was the life of rats infected by flies, *i.e.* the greater was the virulence of the trypanosomes (his rats lived on the average 18 days). Burtt points out that though Vanderplank's suggestion is borne out by the short duration of the infection in rats, it is not supported by the low transmissibility in the flies. In the experiments conducted by Burtt the temperature of the antbear was 95°F., the percentage of flies having salivary gland infections was 1·5, while the average duration of life of rats infected by the flies was 40·5 days. These results agree with those of Vanderplank as regards the low infection rate in the flies, but they do not support his contention that transmissibility and virulence of *T. rhodesiense* may be increased by passage through hosts with a low mean body temperature.

C. A. Hoare.

FULTON, J. D. & STEVENS, T. S. **The Glucose Metabolism** *in vitro* **of** *Trypanosoma rhodesiense.* *Biochem. J.* 1945, v. 39, No. 4, 317–20. [23 refs.]

The authors have studied the glucose metabolism of *Trypanosoma rhodesiense* maintained in a medium containing a small amount of serum. Some tests were conducted in a serum-free medium, but the trypanosomes did not survive so well, with the result that the yield of metabolic products was low. The products isolated were succinic, pyruvic, lactic, acetic and formic acids, glycerol, ethanol and carbon dioxide. The method of detection of these products is described and the possible paths of their production, and the results of earlier work on trypanosome metabolism, are discussed. *C. M. Wenyon.*

BURTT, E. **Salivation by** *Glossina morsitans* **on to Glass Slides: a Technique for isolating Infected Flies.** *Ann. Trop. Med. & Parasit.* 1946, July, v. 40, No. 2, 141–4, 10 figs. on 2 pls.

The author has developed a discovery originally made by BRUCE, that tsetse can be made to salivate on a cover slip. If the fly has a salivary infection the trypanosomes are deposited and may be stained and studied. The chance of finding them is increased if the cover slip is smeared with egg albumin.

Burtt finds that the method is a relatively quick and simple way of identifying those individual flies with a salivary infection of *T. rhodesiense.* The method, if repeated on several days, reveals infections which are missed if each fly is fed on a separate rat (which must then be isolated and observed over a period, a laborious method).

Burtt has observed bacteria in saliva, in a very small proportion of flies. The bacteria (which are of several morphological types) may occur in the presence or absence of trypanosomes, though significantly more frequent in their presence. The bacterial infection in the fly is persistent, but apparently harmless.

P. A. Buxton.

CHABEUF, M. & MBAPPE, J. Les "grands suspects" de trypanosomiase dans les environs d'Ebolowa (Cameroun). [**Suspected Sleeping Sickness Cases in the Region of Ebolowa, French Cameroons.**] *Bull. Soc. Path. Exot.* 1946, v. 39, Nos. 5/6, 188–93.

Sleeping sickness does not occur in the Ebolowa subdivision to the same extent as in other parts of the French Cameroons, although it has been of serious consequence for the past 20 years in the Nyong Valley, only 100 km. away. In 1942 no more than 212 persons were found to be infected among 45,000 examined in the subdivision, and three peculiarities were noted at that time :—(1) the occurrence of cases clinically resembling sleeping sickness, but in which trypanosomes could not be found in the blood, gland juice or c.s.f. ; (2) extreme paucity of tsetse fly ; and (3) the high proportion of cases whose infections were acquired in other regions.

It is not uncommon to find patients whose c.s.f. is grossly abnormal, and the question repeatedly arises whether such cases, in which the patients are suspected of suffering from sleeping sickness, are not in fact cases of syphilis. In order to investigate this problem the authors decided to keep under observation in the Ebolowa Hospital, for one year, 26 patients suffering from chronic nervous (and in most cases mental) disturbances. Only two of these patients came from outside the region.

Examination of the blood, even after provocative injection of sodium thiosulphate or other substances, consistently failed to reveal trypanosomes, except in one patient, who had been ill for three years and whose c.s.f. was grossly pathological. Bone marrow examinations revealed no trypanosomes in any case.

In 18 of the patients the cell-count and albumin-content of the c.s.f. were markedly increased, but this picture improved (though only for a month or two) after tryparsamide, in the 13 cases so treated. The authors consider this [with doubtful justification] to be strongly suggestive of sleeping sickness as the correct diagnosis.

The Vernes reaction, which is ordinarily negative in trypanosomiasis uncomplicated by syphilis, was positive in only three out of eight cases suspected of being syphilitic.

The most interesting clinical feature of the entire group was the frequency of hemiplegias. This was the presenting condition in 12 of the cases. It was of sudden onset, together with fever and headache, and tended to improve in most cases without any treatment, but leaving the patient, after several months, with characteristic residual impairments of movement, speech, and ideation. The other clinical signs and symptoms of these and the remaining cases are fully described, and the authors conclude that the variety of signs, and the impossibility of systematizing them, can only lead to the conclusion that they represent more or less intense and irreversible disseminated lesions, attributable to a trypanosomal encephalitis, possibly due to the well-known diffuse perivascular infiltration of sleeping sickness.

Nearly all the cases which most strongly suggested nervous trypanosomiasis responded rapidly to tryparsamide, but relapses were frequent, and some such patients died, to all appearances as cases of sleeping sickness. In the absence of specific treatment the patients progressively deteriorated. Cases in which the c.s.f. was normal, or nearly normal, were especially refractory to treatment, and anti-syphilitic treatment hardly ever gave encouraging results.

The authors feel justified in diagnosing sleeping sickness in most of these cases, on general clinical grounds, as well as by a process of exclusion of other diseases. The epidemiological peculiarity is that in a region free from endemic sleeping sickness there are always to be found a small number of cases

of this disease in which trypanosomes cannot be seen. Where, when and how did these persons become infected ? The authors discuss various possibilities, without reaching any definite conclusions.

The clinical differentiation between trypanosomiasis and syphilis is not always as clear-cut as is often supposed. The fact that classical tabes and G.P.I. are still unknown in the area under review does not mean that central nervous system syphilis does not occur. Syphilitic myelitis, cerebral arteritis and meningitis undoubtedly do occur, though rarely, and four of the present series were found to be syphilitic. The diagnosis of syphilis is, however, often reached too hastily. If one carries out serological tests among indigenous patients formerly given insufficient treatment on a diagnosis of syphilis, one rarely finds unequivocal positive reactions coinciding with nervous troubles characteristic of that disease.

The authors discuss other possible causes of the conditions they have observed. They write that the neurotropism of tropical infectious diseases is as yet insufficiently explored, and they admit that their own incursion into the subject is to present, rather than to solve, a problem which remains for further investigation. *E. M. Lourie.*

BELL, F. R. & JONES, E. R. **Carbohydrate Metabolism in Bovine Trypanosomiasis.** *Ann. Trop. Med. & Parasit.* 1946, July, v. 40, No. 2, 199–208, 3 figs. [11 refs.]

CARINI, A. Divagações otimístas sobre o porvir de doença de Chagas na América do Sul. [**Observations on the Future Significance of Chagas's Disease in South America.**] Reprinted from *Arquivos de Biologia.* 1943, Sept.–Oct., v. 27, No. 257, 4 pp.

ROMEIRO, O. dos S. Mais dois casos agudos de doença de Chagas constatados em Pires do Rio, Estado de Goiaz. [**A Report on Two Cases of Chagas's Disease in Pires do Rio.**] *Brasil-Medico.* 1946, July 20 & 27, v. 60, Nos. 29/30, 243–5, 2 figs.

MAGALHÃES, B. F. & FREIRE, S. A. Um aspecto electrocardiográfico da doença de Chagas. [**The Electrocardiographic Aspect of Chagas's Disease.**] *Mem. Inst. Oswaldo Cruz.* 1945, Oct., v. 43, No. 2, 287–99, 7 pls. [31 refs.] English summary (8 lines).

RANDOLPH, N. M. **DDT for the Control of** *Triatoma.* *J. Econom. Entom.* 1946, June, v. 39, No. 3, 419.

FULTON, J. D. & HARRISON, C. V. **An Outbreak of** *Trypanosoma cruzi* **Infection in Indian Monkeys.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1946, June, v. 39, No. 6, 513–20, 6 figs. on 1 pl. [11 refs.]

The authors report the occurrence in Liverpool of infection with *Trypanosoma cruzi* in six macaque monkeys out of a batch of ten recently imported from India, and in two others which had been in the laboratory for two years. The trypanosome had all the morphological, developmental and cultural characters of *T. cruzi*, which are described in detail. In connexion with experimental work carried out on these monkeys, it was possible that in some cases the infection was transmitted from one monkey to another by blood inoculation.

In discussing the possible source of these laboratory infections the authors note :—(1) that the ship in which the monkeys had been transported called at a number of ports in South America and passed through the Panama Canal, *i.e.* they had been in the endemic areas of Chagas's disease ; (2) that a strain of

T. cruzi in mice is kept in the same building ; and (3) that bed-bugs were also discovered there, but none was found in the monkey cages, and those examined for flagellates were negative. Infection with *T. cruzi* had already been reported from Asiatic monkeys kept in European and North American laboratories on several previous occasions.

To account for such laboratory infections some observers have suggested that the distribution of *T. cruzi* is not restricted to the New World, and that the infections in the monkeys had been naturally acquired by them in Asia. The present authors leave open the question regarding the source of infection in their monkeys, but do not exclude the possibility of a laboratory infection acquired in Britain.

[Though the fact is not mentioned in the present paper, the reviewer is aware that in at least two of the laboratories to which the authors refer, strains of *T. cruzi* are maintained in rodents, while in one of them a search, carried out after the discovery of *T. cruzi* infection in monkeys, revealed the presence of bed-bugs in the animal house. Thus in all these cases of laboratory infection in monkeys the epidemiological factors for the transmission of the infection from the rodents to the monkeys seem to have been present.] The authors conclude that "the possibility of *T. cruzi* infection should be borne in mind when working with rhesus monkeys." [This warning might be extended by pointing to the potential danger of the infection being transmitted to human beings by bed-bugs under similar conditions.] *C. A. Hoare.*

FRIEDHEIM, E. A. H. & BERMAN, Rose L. **An Organic Antimony Compound with Curative and Prophylactic Activity in Experimental Trypanosomiasis.** *Proc. Soc. Exper. Biol. & Med.* 1946, June, v. 62, No. 2, 131–2.

Two types of compound, both metal-free, are known to have a prophylactic effect in trypanosomiasis : (i) the polysulphonated ureide Suramin (Bayer 205, Antrypol, etc.) ; and (ii) amidines, such as Pentamidine and Stilbamidine. A third type, a quinquevalent antimonial, is now described. It is a polymerized sodium salt derived from *p*-(2,4-diamino-1,3,5-triazinyl-6) aminophenylstibonic acid (synonym : melaminylphenylstibonic acid).

In mice this water-soluble compound is tolerated at a single intraperitoneal dose of 2·5 gm./kgm., and *T. equiperdum* infections are cured by 0·0125 gm./kgm., the therapeutic index therefore being 200.

Two experiments are described to illustrate the prophylactic property of the compound. Five mice were treated intraperitoneally by 0·05 gm./kgm. (*i.e.* 1/50th of the 100 per cent. tolerated dose), and were inoculated 69 days later with *T. equiperdum*. They all remained uninfected. The mechanism of this prophylactic action seems to be based on the fact that the drug is resorbed and eliminated so slowly that a sufficient trypanocidal concentration is maintained in the organism over a considerable length of time. This theory is substantiated by the fact that the *p*K of the compound is such that the free stibonic acid is precipitated by CO_2 from solutions of its sodium salt, and after intraperitoneal and intramuscular injections of the salt a white deposit of the free acid can be readily seen for many weeks on the surface of the peritoneum and between the muscle fibres.

The second experiment illustrates prophylaxis against repeated inoculation. A single intraperitoneal injection of 0·3 gm./kgm. was administered to 105 mice. One month later they were inoculated with *T. equiperdum*. None became infected. The test-inoculation was then repeated at weekly intervals. All the mice remained free of infection for 50 days from the start of the experiment, but infections began to appear thereafter, so that by the 100th day about half, and by the 164th day all, the mice had exhibited the infection. When this did

eventually arise it differed basically from the classical fulminating septicaemic type, and was of a chronic nature, such as occurs in man and rabbits, with parasites appearing and vanishing from the blood at irregular intervals. In this experiment the mechanism of prophylaxis possibly included an immunological factor. *E. M. Lourie.*

MAYER, R. L. & BROUSSEAU, Dorothy. **Development of Immunity to Reinfection during Chemoprophylaxis of Trypanosomiasis with a New Antimony Derivative.** *Proc. Soc. Exper. Biol. & Med.* 1946, June, v. 62, No. 2, 238–40.

The authors have confirmed the claim of FRIEDHEIM and BERMAN [above] that mice treated with a new antimonial, *p*-(2,4-diamino-1,3,5-triazinyl-6,) aminophenylstibonic acid, are protected from infection by *T. equiperdum*, repeatedly inoculated over long periods.

Mice treated by a single dose of 0·05 gm./kgm. [route not stated], and subsequently inoculated and reinoculated up to 45 times with *T. equiperdum*, remained uninfected for periods up to 330 days, but did eventually become susceptible to infection. Such protection, lasting two months or more, also arose after oral administration of 0·025 or 0·050 gm./kgm. on five successive days.

Two series of experiments, as follows, were performed in order to show the possible rôle of immunological processes in the protection produced by these means :—

1. Normal uninfected mice were treated subcutaneously by 0·05 gm./kgm. Three weeks later serum obtained from these animals was pooled and injected into normal mice, which were then inoculated with trypanosomes ; typical fatal infections resulted. The experiment was repeated in exactly the same way, except that the initially drug-treated mice had been infected with trypanosomes 48 hours previously ; contrary to the experience with the other experiment, the serum obtained three weeks later from these mice was found to protect a further series of mice from infection. Other such experiments showed that the anti-trypanocidal [*sic*] effect was obtained five days after the treatment of infected mice. These experiments show that the trypanocidal power of the serum of infected mice, treated by the antimonial, is not necessarily due to the drug persisting in the body, but possibly to other, probably immunological, processes. Similar results are obtained by the use of neoarsphenamine instead of the antimonial.

2. The development of this property of the mouse-blood is probably an effect of successful treatment and subsequent death of the parasites, as has been shown by much other previously published work. In support of this, the authors found that mice inoculated with trypanosomes (*T. equiperdum*), which had been killed by repeated freezing and thawing, were partially protected against infection, the protection being manifested by a protracted incubation period on subsequent inoculation of viable trypanosomes. *E. M. Lourie.*

LEISHMANIASIS.

GHOSH, H., GHOSH, N. N. & RAY, J. C. **A Preliminary Note on the Complement-Fixation Reaction in Kala-Azar with a Specific Antigen as an Aid to Diagnosis.** *Ann. Biochem. & Exper. Med.* Calcutta. 1945, v. 5, No. 4, 153–8. 11 refs.]

The authors report very successful results in diagnosis of kala azar by the complement fixation reaction, in which an antigen prepared as follows was used.

Cultures of *L. donovani* on Ray's medium [this *Bulletin*, 1933, v. 30, 322] were emulsified with 0·25 per cent. phenol in distilled water. The emulsion, matched with No. 8 of Brown's opacity tubes, showed 80 million flagellates per cc. The emulsion was kept for 25 days in a glass phial in the incubator at 37°C., being taken out each day to undergo mechanical shaking for two hours. At the end of the period the contents of the phial were centrifuged, the supernatant fluid, after filtration, being used as the antigen. The test was carried out with slight modification according to Method 4 of the Medical Research Committee's Report on the Wassermann test (1918). It was found to be highly specific, being positive in early cases of kala azar when the usual serum tests are still negative. *C. M. Wenyon.*

COLLIER, H. O. J. & LOURIE, E. M. **The Action** *in vitro* **of Diamidines and other Compounds on** *Leishmania donovani*. *Ann. Trop. Med. & Parasit.* 1946, Apr., v. 40, No. 1, 88–100. [25 refs.]

The culture medium employed in the tests described was composed of one part of fresh unheated rabbit serum mixed with two parts of a 12·5 per cent. solution of rabbit red blood corpuscles in isotonic saline. The solution was prepared by taking one volume of defibrinated rabbit blood with six volumes of sterile distilled water, adding one volume of 6·3 per cent. sodium chloride solution, centrifuging and discarding the deposit. As the highest temperature at which satisfactory growth of *L. donovani* was obtained was 34°C. it was at this temperature that the tests were carried out. A series of tubes of medium containing two-fold dilutions of a drug to be tested were inoculated with flagellates. The tubes were examined on the fifth day and the lowest concentration of drug giving a negative tube was regarded as the effective dose. A total of 40 compounds, mostly aromatic diamidines or closely related substances, were tested. The only ones showing considerable activity were some, but not all, of the diamidines. Antimonials of known activity *in vivo*, such as tartar emetic and neostam, were relatively inactive. As regards the diamidines, there was a fair correlation between activity *in vitro* and the reported activity *in vivo*. Compounds with an *in vitro* titre of one million or more had been found to be effective *in vivo*. The *in vitro* technique would appear to be of some value as a screening procedure, at least in this particular chemical group. The leishmanicidal activity of the diamidines has been studied from the point of view of chemical constitution, and compared with the similar study of EWINS and his colleagues in 1942 on trypanocidal activity [*Jl. Chem. Soc.*, 1942, 103]. *C. M. Wenyon.*

KAMEL, A. **Leishmaniasis of Eyelid.** *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 124–8.

Cutaneous leishmaniasis is rarely met in ophthalmology; the author describes three cases. The first had a warty growth along the whole length of the left lower eyelid. The second had a small papule in the centre of the left upper eyelid just above the eyelashes, which gradually increased in size and ulcerated. The margins became undermined, but not indurated, and the ulcer was not adherent to the tarsus. The third had an ulcer with a granular surface, situated on the left side of the nose and cheek. These cases occurred in localities in which the disease has not previously been seen.

Different forms of treatment are mentioned, of which X-ray treatment and diathermy are said to be the best. [No mention is made of intravenous injections of antimony in the general treatment of the disease.]

E. O'G. Kirwan.

MÉCHIN, R. Quatre nouveaux cas de bouton d'Orient du Tell constantinois traités par la quinacrine locale. [**Quinacrine Treatment of Four New Cases of Oriental Sore in the Constantine Region.**] *Arch. Inst. Pasteur d'Algérie.* 1946, Mar., v. 24, No. 1, 57–9.

This is a record of four cases of oriental sore from localities in the neighbourhood of Constantine in Algeria. They are of interest in that they are outside the Sahara area, where the disease is more frequently encountered. The four cases were treated with quinacrine (atebrin) injected into the sores, all of which were on the face. For sores not too near the delicate tissues of the eyelids 0·3 gm. of the drug is injected once or twice. For sores on the lids not more than a third of this quantity is injected at one sitting. When the full dose is given healing is rapid, cicatrization being complete in three or four weeks. In one case after healing an oedema in the sub-orbital region persisted. This is attributed to the toxic action of the quinacrine on the soft tissues in that neighbourhood.

C. M. Wenyon.

PANJA, G. **Treatment of Oriental Sore by X-Rays.** *Indian Med. Gaz.* 1946, June–July, v. 81, Nos. 6/7, 251.

ANSARI, M. N. Culture et isolement de *Leishmania tropica.* Leishmanisation prophylactique. [**Culture of *L. tropica* for Prophylactic Inoculation.**] *Arch. Inst. d'Hessarek.* Teheran. 1946, Jan., v. 2, No. 2, 31–5.

Working in Teheran, the author describes his technique for obtaining cultures of *Leishmania tropica.* With these cultures he has inoculated 120 persons by scarification or intradermally with a view to protecting them against naturally-acquired disfiguring sores on the face. In 90 per cent. of the cases, sores developed at the site of inoculation within 20 days to 7 months. As 85 per cent. of the cases of oriental sore are in children in their tenth year it would appear that protective inoculations should be carried out before this age.

C. M. Wenyon.

LEITÃO, J. S. "Ponos" canina. [**Canine Leishmaniasis in Portugal.**] *Rev. Med. Vet.* Lisbon. 1945, v. 40, 229–43. English summary. [Summary taken from *Vet. Bull.* 1946. July, v. 16, No. 7, 218. Signed U. F. RICHARDSON.]

Attention is drawn to the prevalence of canine leishmaniasis in Portugal. In Lisbon it has been estimated that 3–4 per cent. of dogs are infected. As a check on the value of diagnostic methods, a full investigation was made of a clinical case of leishmaniasis in a dog from Coimbra. Positive results were obtained by examination of smears of tibial bone marrow and of the contents of cutaneous ulcers, and the formol-gel and Takata-Ara reactions were positive. Blood, liver, skin and ungual matrix smears were negative, as were serum-flocculation and the neostibosan and formol neostibosan reactions.

PESSÔA, S. B. & BARRETTO, M. P. Sôbre a localização dos parasitas nos tecidos e a intensidade do parasitismo na leishmaniose tegumentar americana. [**Distribution of Parasites and Intensity of Parasitism in S. American Cutaneous Leishmaniasis.**] Reprinted from *1 Reunião Anual dos Dérmato-Sifilógrafos Brasileiros, Rio de Janeiro, 26–28 Sept., 1944.* 1945, 123–36, 8 figs, on 4 pls. [31 refs.]

The authors discuss the distribution of leishmania in the tissues of patients with S. American cutaneous leishmaniasis, and the variations in intensity of the infections. They show that the parasite is nearly always intracellular, but may

lie extracellularly between cells which have been separated by oedema resulting from subjacent inflammation. The type of cell most commonly infected is the histiocyte, but parasites may occur in many other types of cells, including polynuclear leucocytes and fibroblasts. The authors were not able to confirm the statements that parasites may occur in lymphocytes, eosinophiles or epithelial cells. As regards the intensity of infections, the leishmania are most numerous in the early lesions which are developing rapidly, particularly at the margins of the papule. When ulceration commences, parasites may still be numerous before secondary bacterial contamination becomes heavy. In old chronic lesions with intense bacterial invasion it may be difficult or impossible to find them. They also occur in small numbers in the lymphatic glands adjacent to the cutaneous lesions and in the lymphatics leading from them, especially in the nodes which develop along their course. When mucosal lesions appear, the parasites are again numerous in the early stages. They become increasingly difficult to find as the lesions become old and when they are secondarily contaminated. *C. M. Wenyon.*

PESSÔA, S. B. & BARRETTO, M. P. Sôbre as lesões iniciais na leishmaniose tegumentar americana. [**The Initial Lesions in S. American Cutaneous Leishmaniasis.**] Reprinted from *1 Reunião Anual dos Dérmato-Sifilógrafos Brasileiros, Rio de Janeiro, 26–28 Sept., 1944.* 1945, 27–35, 9 figs. on 3 pls. [22 refs.]

The initial lesion in S. American cutaneous leishmaniasis is shown to be a papule or nodule, which may or may not be pruriginous or painful. Not infrequently there is an accompanying adenitis, giving rise to a combined primary infection of the skin and glands. In these primary lesions parasites are usually very numerous. The paper is illustrated by a series of photographs showing the primary lesions. *C. M. Wenyon.*

MILLAN GUTIERREZ, J. Leishmaniasis de la piel y de las mucosas. Información General. [**Cutaneous and Muco-cutaneous Leishmaniasis.**] *Medicina.* Mexico. 1946, Apr. 25, v. 26, No. 506, 150–59, 6 figs.

A general account of cutaneous and muco-cutaneous leishmaniasis of Central and South America, intended presumably for the information of local medical men. *C. M. Wenyon.*

IRIARTE, D. R. Contribución al estudio de la leishmaniosis en Venezuela. [**Leishmaniasis in Venezuela.**] *Bol. Laboratorio Clinica "Luis Razetti."* 1946, June, v. 6, Nos. 19/20, 355–93, 16 figs. [Bibliography.] English summary.

This is an illustrated account of muco-cutaneous leishmaniasis in Venezuela with special reference to cases having involvement of the nose, mouth and pharynx. Short histories are given of 13 such cases in patients from 15 to 57 years of age. In the nose the destructive process involves the soft parts and cartilage of the septum, leading to depression of the tip of the nose, producing a characteristic parrot-beak appearance. In the mouth and pharynx the ulceration of the mucosa leads to a thickening of the lips and other affected parts. The chief inconvenience complained of by the patients is nasal obstruction, for, unlike other conditions involving the nose, such as syphilis or tuberculosis, there is little or no discharge. As a rule leishmania can be discovered in smears made from the lesions if patient search is made. Thus they were found in all but one of the cases described. When parasites cannot be discovered, the intradermal test of Montenegro is useful in confirming a clinical

diagnosis. Treatment has been by intravenous injection of tartar emetic. Certain patients have tolerated daily doses of 0·1 gm. till a total of 2 gm. has been given, but actually 0·08 gm. is the highest single dose that can be administered with safety. Under this treatment there has been improvement, but not complete cicatrization of the mucosal lesions. In connexion with the transmission of the disease, five species of *Phlebotomus* are mentioned as possible vectors. In two (*P. longipalpis* and *P. migonei*) previous investigators have recorded leptomonad infections. *C. M. Wenyon.*

CERRUTI, H. A reação intradérmica de Montenegro nas cicatrizes inespecíficas dos leishmanióticos. [**The Montenegro Reaction in Non-Specific Scars of Leishmania Patients.**] *Arquivos Dermat. e Sifiligrafia de São Paulo.* 1945, Sept., v. 9, No. 3, 71–9. [22 refs.]

In twenty-five cases of cutaneous leishmaniasis in Brazil the author has carried out the Montenegro test on the normal skin, the scars of healed leishmania lesions, the scars of smallpox vaccination, the scars resulting from previous Montenegro tests and scars resulting from other causes. All the tests were positive, indicating that the specificity of the test is not influenced by the cicatricial changes in the skin. *C. M. Wenyon.*

FEVERS OF THE TYPHUS GROUP.

LOWE, J. **Ten Cases of Typhus in Civilians in Calcutta.** *Indian Med. Gaz.* 1946, Apr.–May, v. 81, Nos. 4/5, 171–4.

The author briefly describes ten cases of fever of the typhus group seen by him in the last quarter of 1945 at the Calcutta School of Tropical Medicine. Only one case had previously been seen at the school—in 1944—although a sharp look-out had been kept since 1942 because of the known occurrence of cases among British and Indian troops stationed in and near Calcutta.

The diagnosis was based chiefly on the serum-agglutination reactions, according to which four were diagnosed as scrub typhus, four as murine typhus, one as tick typhus, and the remaining case, the only one that was fatal, as of doubtful type. A petechial rash was seen in two cases. No mention is made of any attempt to recover the causative organism.

Eight of the patients had lived in the heart of Calcutta ; one of these had gone outside the city on fishing excursions, another had made a journey to Bombay, the rest were not known to have left their homes [but the serological groups to which these, or the two patients from rural areas, belonged, are not distinguished]. No local eschar was seen, In two cases there was slight general enlargement of the lymph nodes.

No further cases were seen till May 1946, when one patient was admitted for scrub typhus. *John W. D. Megaw.*

HABIBI, M. Etude des lésions anatomo-pathologiques du typhus exanthématique au cours de l'épidémie de 1942–1943 en Iran. [**Study of the Pathological Anatomy of Exanthematic Typhus in the Epidemic of 1942–1943 in Iran.**] *Arch. Inst. d'Hessarek.* Teheran. 1946, May, No. 4, 63–75, 12 figs. on 2 folding pls. [25 refs.]

The opportunity for this study occurred in Iran, where in the months January to July 1943 there were 15,500 cases of typhus fever, with a fatality rate of 17 to 20 per cent.

The naked-eye and microscopical lesions are clearly described; the latter are illustrated by 12 reproductions of photomicrographs. The changes observed were of the usual types.

The author was not satisfied with the results obtained by the Castañeda and other standard methods of staining; he recommends prolonged staining (24–48 hours) with Giemsa's stain and differentiation with a mixture of absolute alcohol, nine parts, and oil of cloves, one part. *John W. D. Megaw.*

CODELEONCINI, E. Le reazioni di Widal e Weil-Felix nella practica sull' altopiano Etiopico. [**The Widal and Weil-Felix Reactions in the Abyssinian Highlands.**] *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1945, v. 5, Nos. 5/6, 71–84. English summary.

The author found that serum retained its agglutinating properties much longer when separated from the clot as early as possible.

He points out that serum when exposed to a hot and dry atmosphere without being sealed up may become so concentrated that the agglutination titre is liable to be considerably increased. He found that when dried clot was the only material available, this, when reconstituted with serum, yielded titres about one-tenth as high as the original serum. *John W. D. Megaw.*

D'IGNAZIO, C. & CODELEONCINI, E. La reazione di Weigl nel tifio esantematico. [**The Weigl Reaction in Typhus Fever.**] *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1945, v. 5, Nos. 5/6, 199–210. English summary (6 lines).

The authors carried out 7,500 rickettsia-agglutination tests on the lines originally worked out by WEIGL; presumably the rickettsial suspensions were prepared from the guts of infected lice.

Between 1938 and 1945 a series of 2,650 Weigl tests were performed simultaneously with Weil-Felix tests; 1,640 of these were of sera from 368 cases of louse-borne typhus, the rest included sera of typhus convalescents, healthy persons (vaccinated and unvaccinated), trachoma patients, Abyssinian prisoners, and persons employed in the laboratory as donors of blood meals for infected or uninfected lice.

The conclusions reached were that although the Weil-Felix test was highly reliable, the Weigl test had the advantages of never becoming positive in patients suffering from diseases other than typhus, and of yielding a significant response about one day earlier.

Apart from the earlier rise and an earlier fall in the Weigl titre, the agglutination curves of the two reactions were parallel, though the Weigl curve was at a lower level. *John W. D. Megaw.*

D'IGNAZIO, C. Osservazioni e considerazioni sulla terapia dei dermotifosi. [**Observations and Discussion on the Treatment of Typhus Fever.**] *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1945, v. 5, Nos. 5/6, 29–50. English summary (4 lines).

Among the many special forms of treatment tried by the author without obvious success were :—convalescent serum, autohaemotherapy, specific immune serum, colloidal carbon intravenously, congo red and cyanide of mercury, mercurochrome, and vaccine therapy. A line of treatment which included intravenous injection of vitamin C and intramuscular injection of vitamin B_1 was tried in about 300 cases in Addis Ababa, and the author claims that this caused an obvious improvement in the general condition of the patients and that

the fatality rate was reduced from 24–27 per cent. to 12–13 per cent. No absolute claim is made for the efficacy of the treatment, but the method was found safe and satisfactory. [See also GIUNTA and D'IGNAZIO, this *Bulletin*, 1939, v. 36, 989.] *John W. D. Megaw.*

D'IGNAZIO, C. & CODELEONCINI, E. Le vaccinazioni contro il tifo esantematico in Etiopia con il vaccino di Weigl dal 1938 al 1945. [**Vaccination against Typhus Exanthematicus by Weigl's Vaccine in Abyssinia from 1938 to 1945.**] *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1945, v. 5, Nos. 5/6, 51–70. English summary (4 lines).

About 40,000 persons were vaccinated in Addis Ababa with a vaccine, of which 16,000 doses were obtained from Weigl's laboratory, and about 25,000 were prepared at Addis Ababa by workers who were personally instructed by Weigl. The incidence of the disease was greatly reduced among the vaccinated as compared with unvaccinated persons exposed to similar risks, though it was suspected that many sub-clinical attacks occurred among the vaccinated. In the cases that were diagnosed the symptoms were exceedingly mild and few complications occurred.

Owing to shortage of the vaccine, trials were made of doses reduced to one-half, one-quarter, one-eighth, and even one-sixteenth of the usual amount. Among 10,000 persons vaccinated with these small doses the results did not appear to be materially different from those obtained with full doses.

John W. D. Megaw.

CODELEONCINI, E. Sulla vaccinazione con escrementi di pidocchio infetto di tifo epidemico. [**Vaccination with the Faeces of Lice infected with Typhus Exanthematicus.**] *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1945, v. 5, Nos. 5/6, 221–9. English summary (2 lines).

The paper contains a description in general terms of the preparation of a vaccine from the faeces of lice infected with louse-borne typhus. The vaccine has been found effective in immunizing experimental animals and good results are claimed from the use of the vaccine on 5,000 persons. Anaphylactic disturbances are said to be thrice as frequent as with the standard Weigl vaccine, which itself is regarded by most workers as obsolete because of the difficulty and cost of production. *John W. D. Megaw.*

SMITH, P. K. **The Use of Para-Aminobenzoic Acid in Endemic (Murine) Typhus.** *J. Amer. Med. Ass.* 1946, Aug. 3, v. 131, No. 14, 1114–17, 1 chart. [Refs. in footnotes.]

Para-aminobenzoic acid was given to 29 patients suffering from murine typhus; the dosage was 2·0 gm. given every two hours with bicarbonate of soda, till the temperature fell to normal. A similar number of cases, "roughly comparable", served as controls. The average duration of the fever in the treated was 10·2 days, and in the controls 13·2 days. There was one death in each group. The treated patient who died was 78 years old; the drug was started on the 10th day and was continued for four days till the temperature became normal, but the patient died of cardiac failure on the 18th day. Penicillin had been given throughout the illness from the 5th day, in doses of 20,000 units every three hours, and sulphadiazine was given in doses of 1·0 gm. every four hours from the 5th to the 10th day.

The author suggests that sulphonamides may be harmful; he did not regard penicillin as having been helpful.

Caution is advised in cases of kidney or liver damage, though no toxic effects were observed from the use of the drug in the present series or in 17 cases of other diseases in which the average total dose was 95 gm. Further trials under more rigorous control are recommended. *John W. D. Megaw.*

MACKIE, T. T. *et al.* **Observations on Tsutsugamushi Disease (Scrub Typhus) in Assam and Burma. Preliminary Report.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1946, Aug., v. 40, No. 1, 15–46, 3 figs. [39 refs.] Discussion 46–56, 2 graphs. [MELLANBY, K. ; SHORTT, H. E. ; SAYERS, M. H. P. ; FAIRLEY, N. H. ; FELIX, A. ; CLARK, R. H. P. ; HOLLINS, C. ; MACKIE (in reply).]

This paper is essentially similar to the one by the same author and 13 collaborators already reviewed [see this *Bulletin*, 1946, v. 43, 917]. The larger and more detailed maps illustrating the present paper are very helpful.

Some interesting points arose in the discussion following the reading of the paper. Dr. Kenneth MELLANBY said that after one negative experiment he had been able to confirm the observation that partially fed mites, when removed from their hosts, reattached themselves to another host in considerable numbers; he added that badly conducted anti-rat measures might sometimes increase the risk of infection. He mentioned that benzyl benzoate, as shown by later work, might be more protective than dibutyl phthalate.

Col. H. E. SHORTT enquired whether the author's findings had thrown any light on the occurrence of different serological types in India, and mentioned that among 65 Weil-Felix-positive sera tested by himself in Madras, 42 were of the *OX19* type, 16 of the *OXK* type, and 7 of the *OX2* type. The reply was that although a few atypical reactions had been observed there was no evidence of the occurrence of other forms of typhus in the area investigated.

Lt.-Col. M. H. P. SAYERS showed two graphs which clearly demonstrated that the disease was definitely seasonal in south-east Asia. The curves of incidence among the troops in 1943, 1944, and 1945 showed a considerable rise in June or July, reaching the maximum in August or September, gradually falling to a low level by January or February, and remaining low throughout February, March, April, and May, though the season of greatest operational activity was the first half of each year.

Brigadier Hamilton FAIRLEY told how dimethyl phthalate had been found by the Orlando group of entomologists in the U.S.A. to be the best of the known protectives against mites, and to be a very effective repellent of mosquitoes ; how this had been adopted on a large scale by Australian forces in 1943, and how it came to be replaced by dibutyl phthalate which, however, was less repellent to mosquitoes.

Dr. A. FELIX stressed the importance of the wild rat as a reservoir of infection and expressed the view that an anti-rat campaign might be even more useful in peace-time than anti-mite measures ; he quoted R. R. PARKER as an advocate of the opinion that susceptible wild animals were essential to the maintenance of infection in the case of the analogous disease, Rocky Mountain spotted fever.

In replying, Col. MACKIE did not dispute the evidence of a special seasonal incidence of the disease, but emphasized the fact that the disease could occur at any season ; he also disavowed any intention of minimizing the importance of rodent reservoirs of infection. *John W. D. Megaw.*

NOAD, K. B. **Tsutsugamushi Fever in Natives.** *Med. J. Australia.* 1946, July 6, v. 2, No. 1, 20–21.

Details are given of two cases in which tsutsugamushi disease occurred in natives of New Guinea. The *Proteus OXK* titre rose to 1–840 in one case and

to 1–1,280 in the other. Clinically the attacks were entirely in keeping with the diagnosis. Another case was probably of the same kind but it was incompletely observed.

The author [reasonably] suggests that the supposed immunity of natives in affected areas is due to previous attacks. *John W. D. Megaw.*

LIKOFF, W. **Changes in the Cardiovascular System in Scrub Typhus in Early Convalescence.** *Amer. J. Med. Sci.* 1946, June, v. 211, No. 6, 694–700, 4 figs.

A group of 100 patients whose ages ranged from 19 to 35 years was studied after an average interval of 49 days from the onset of scrub typhus. The febrile period had lasted only nine days on the average. All but one of the patients had been ambulatory for at least 14 days before examination.

Among 30 patients who complained of symptoms of rapid heart beat or other cardiovascular disturbance the pulse rate was over 100 in all but two. Among the other 70 only four had a rate of 100 or more.

An unselected group of 25 patients were tested for exercise-tolerance ; 12 of these had a pulse rate which remained 10 beats higher after a resting period of two minutes, and only one of the 12 had not complained of cardiovascular symptoms.

Electrocardiographic abnormalities were found in 10 patients, all of whom had complained of symptoms. The chief changes found were :—bundle-branch block (2) ; intraventricular block (1) ; negative T waves in two or more leads (3) ; splintering of the QRS (3) ; and secondary-degree heart block (1). In five patients the abnormalities persisted till the end of the period of observation ; the most significant changes were in the three patients who had bundle-branch or intraventricular block.

No evidence was found of cardiac failure or organic valvular disease in any patient, though systolic bruits were heard in the pulmonic area in six, and in the mitral area in two. Electrocardiographic investigation is regarded as the most reliable method of estimating myocardial damage. *John W. D. Megaw.*

ROMEO, B. J. **Convalescence from Scrub Typhus.** *Bull. U.S. Army Med. Dept.* 1946, Aug., v. 6, No. 2, 167–73.

A detailed examination was made of 312 patients convalescent from attacks of scrub typhus, many of which had been severe. The general conclusion reached is that every patient who survives the attack will make a complete and permanent recovery.

The patients were seen between July 1942 and September 1944, they had already spent an average period of four weeks in hospitals in forward areas of New Guinea. One patient was suffering from a second attack after an interval of 10 months ; the previous illness had been more severe.

The patients on admission were apprehensive, they expected to be incapacitated for life and anticipated a long period of sick leave. The first step in treatment was to dispel both of these " false beliefs ".

The chief findings on admission were :—an average loss of weight of 15 pounds ; a resting pulse rate of 100 or over in 55 per cent. ; tremors of the outstretched hands in 60 per cent. ; vertigo in 25 per cent. ; general or local adenitis in 98 per cent. ; and pigmentation at the site of the eschar in all the cases, amounting to 70 per cent., in which this lesion had occurred.

After a thorough examination the patients were placed in four different classes according to their fitness for exercise ; only 8 per cent. needed temporary bed treatment. During the stay in the convalescent hospital, which

averaged 28·4 days, all but nine of the patients had been reconditioned and were returned to full duty. Those evacuated for further treatment were suffering from various disabilities unconnected with scrub typhus.

The average period from the onset till discharge to duty was 58·9 days ; it varied from 30 to 133 days. Except for persistent lymph-node enlargement, all the signs and symptoms had disappeared and no electrocardiographic abnormalities remained.

Prolonged rest in bed is strongly deprecated ; the author found that convalescence was greatly hastened by starting active reconditioning treatment as early as possible after defervescence.

[Most observers will agree that the attitude of over-caution and pessimism in connexion with scrub typhus has been responsible for wastage of man-power and injury to the patients, but it must be noted that the fatality rate in the present series of cases had been only 4 per cent. In outbreaks with a fatality rate of 10 per cent. or over the results might not have been so remarkably good.]

John W. D. Megaw.

HICKS, J. D. **Agglutination Titres with** *Bacillus proteus* (*OXK*) **in Human Sera.** *Med. J. Australia.* 1946, July 6, v. 2, No. 1, 19–20.

Sera from four groups of persons were tested for *Proteus OXK* agglutinins :— (*a*) 96 blood donors in Sydney ; (*b*) 100 men from a unit, in New Guinea, in which no cases of scrub typhus had occurred during the previous five months ; (*c*) 104 men from a unit, in the same district of New Guinea, in which 29 cases of scrub typhus had occurred during the previous three months ; and (*d*) 98 natives from a nearby labour camp.

Between 80 and 95 per cent. of the members of all the four groups reacted at titres of 1–20 to 1–40, but in the first two groups only 6 and 3 per cent., respectively, reacted at 1–80, whereas in the last two groups 15 and 17 per cent. reacted at this titre.

The author suggests that these figures may indicate the previous occurrence of infection among the members of the last two groups ; in the case of group C the infection must have been sub-clinical. *John W. D. Megaw.*

BUSTAMANTE, M. E., VARELA, G. & ORTIZ MARIOTTE, C. II. Estudios de fiebre manchada en Mexico. Fiebre manchada en la Laguna. [**Studies in Rocky Mountain Spotted Fever in Mexico.**] *Rev. Inst. Salubridad y Enfermedades Trop.* Mexico. 1946, Mar., v. 7, No. 1, 39–48, 2 graphs & 1 map. [Summary in English by the authors.]

" The authors report the presence of Rocky Mountain Spotted Fever in La Laguna (Coahuila y Durango), Mexico. The epidemiological and experimental studies based on clinical diagnosis, gave as positive results the isolation of two strains of spotted fever virus (western type of the U.S.A.) from two groups of *R. sanguineus* naturally infected.

" One strain was obtained from lot 8, formed by 13 adult *R. sanguineus* gathered on the adobe walls and earth floor of a house in Granada Common ; the second strain was obtained from lot 10 formed by three adult females of *R. sanguineus*, collected on a dog of the Zaragoza Common. The confirmation of natural infection of these ticks from dogs, and now in houses, is important for the epidemiology of Spotted Fever in Mexico and adds *R. sanguineus* to the natural vectors of the disease in the Continent.

" On the same survey were positive the complement fixation tests with sera from two persons recovered from the infection and with sera from two dogs. Lots Nos. 3 and 13 were collected in mattresses where *Rhipicephalus* lived as *Cimex* do in similar conditions.

"The Laguna Region belongs by its geography to the north-central part of the Mexican high plateau which extends to the south-east of United States."

MAZZOTTI, L. & VARELA, G. Conservación experimental del virus de la fiebre manchada en *Ornithodoros furcosus.* [**Experimental Maintenance of Rocky Mountain Spotted Fever Virus in** *Ornithodoros furcosus.*] *Rev. Inst. Salubridad y Enfermedades Trop.* Mexico. 1946, Mar., v. 7, No. 1, 13–15. [Summary in English by the author.]

"A strain of virus of spotted fever isolated from Mexico was maintained experimentally during 345 days in *Ornithodoros furcosus.* This tick transmits the infection through the egg at least to the first generation."

YELLOW FEVER.

DE PAULA SOUZA, G. H. **Fourth Report of the Expert Commission on Quarantine.** *Epidemiological Information Bull.* (UNRRA Health Division). Wash. 1946, July 31, v. 2, No. 14, 580–86.

The Quarantine Commission of the Expert Committee on Health of UNRRA met in New York on July 10th, under the chairmanship of Dr. P. G. STOCK. The main item of interest in their report is an account of the controlled experiment to test the efficacy of the anti-yellow fever vaccine made at the Pasteur Institute at Dakar.

Six hundred French soldiers, none of whom had been outside France or received any inoculation against yellow fever, were divided into three groups of 200 (groups A, B and C). Group A were inoculated by scarification with the Dakar vaccine, group B received the Dakar vaccine plus dry smallpox vaccine, by scarification, and group C received Rockefeller 17D yellow fever vaccine by subcutaneous injection. The inoculations were given on July 4th and 5th, 1945, and blood samples were taken on August 9th, 1945, one-third of each sample being sent to Dakar for testing, one-third to the Montana Laboratory of the U.S.P.H.S. and one-third held in reserve. Ninety-two cases showed discrepancies in degree between Montana and Dakar and the reserve sera were sent for further testing to the Yellow Fever Laboratories in Rio de Janeiro.

Final results showed that 98·94 per cent. of group A, 97·93 per cent. of group B and approximately 65 per cent. of group C had been immunized. [This last figure is calculated from the detailed analysis of results which is shown in a fuller report on the Dakar vaccine printed in *Epidemiological Information Bulletin*, Vol. 2, No. 15, an abstract of which will appear in this *Bulletin*, 1947, Jan., Vol. 44, No. 1.] The results from 30 unvaccinated controls were negative in 29 and inconclusive in 1. The Commission therefore recommended approval of the Dakar vaccine under Article XI (10) of the International Sanitary Convention for Air Navigation of 1944.

The Commission also approved the yellow fever vaccine prepared at the South African Institute for Medical Research, Johannesburg (this is of the Rockefeller type). They approved this Institute and the Pasteur Institute at Dakar under Article XI (11) as Institutes for testing the potency of yellow fever vaccine.

The yellow fever endemic areas in Brazil were somewhat modified and an area 10 kilometres round the town of Asmara in Eritrea was excluded from the endemic area, subject to the monthly *Aëdes* index remaining satisfactory. The questions of differentiating administratively between endemic and epidemic yellow fever areas; of extending the Convention periods for the incubation of

smallpox and typhus; and of the value of the "immunity" reaction following vaccination against smallpox were also discussed, but no decisions were reached. *Neville M. Goodman.*

[In *Epidemiological Information Bulletin*, Vol. 2, No. 16, p. 689, a correction is made to a paragraph in the paper reviewed above, relating to the delineated area in Brazil. This point is not referred to in the above abstract, but the correction reads as follows:—"Attention is called to a printing error in *Epidemiological Information Bulletin*, Vol. II, No. 14, p. 584, line 21: instead of 15°N. read '5°N. parallel of latitude'."—*Ed.*]

BRITISH GUIANA. **Report on the Yellow Fever Control Service of the Medical Department, British Guiana, for the Year 1945, including an Historical Résumé from its Origin to Date** [BEVIER, G., M.D., Chief Officer]. 26 mimeographed pp., with Appendices I & II.

It is known that jungle yellow fever occurs in the inland parts of British Guiana; in 1939 and 1940 SNEATH [this *Bulletin*, 1940, v. 37, 90; 1941, v. 38, 67] reported that nearly 37 per cent. of 506 blood specimens from the interior gave positive protection tests, and one-fifth of these positive specimens were from people under 20 years of age. But the positive specimens were entirely from the interior, and the point is made that although in the past (up to about 1914) the disease occurred in the coastal areas, there is no evidence that it has been endemic there since then. The populated coastal region is separated from the inland jungle area by a wide zone of practically uninhabited country; communication between the interior and the sea is limited to river or air traffic. There is a focus of yellow fever in Venezuela adjacent to the boundary of British Guiana.

The yellow fever control service in British Guiana is concerned most particularly with the control of *Aëdes aegypti* on the coast, especially in the ports and airports; Georgetown is the last port of call for many ships and aircraft leaving the mainland for the islands and countries to the north. One viscerotomy post has been established in the jungle country and others are projected, but in the meantime, mosquito control has been developed.

Aëdes aegypti is common in Georgetown, and control measures have been in force for some years; a trained staff now exists and public co-operation is fairly good. Inspectors visit all houses and have become proficient in detecting breeding places of *Aëdes aegypti*, particularly those likely to be overlooked by the householders—vases, water-containers, roof gutters and the like. In the controlled area there are 29,102 premises. This country receives a heavy rainfall of 80–120 inches.

The most useful index of the prevalence of *Aëdes aegypti* is given by the "premise breeding index", which shows the percentage of premises in which breeding is found; this is based on the reports of inspectors, who make weekly visits to the premises in the area. A group of houses which one inspector can visit between Monday morning and Friday evening constitutes a zone, six zones constitute a District, which is under the control of a Chief Inspector, who is expected to re-inspect about 10 per cent. of the houses in each zone. There are also special services, for distributing larvivorous fish, for dealing with high tanks, and with roof gutters, for oiling drains and other collections of water, and for classification of mosquitoes caught. Squads whose duty it is to capture adult *Aëdes aegypti* have become increasingly important; the most sensitive indication of breeding is the finding of adults in houses, and they can usually be found, if present, by trained inspectors. The roof gutter problem has been costly to solve, but with special training and equipment, periscopes, ladders, etc., a great improvement has been made. A serious complication of this problem is

the fact that eggs of *Aëdes aegypti* can persist in the dry state for over a year, and that they can hatch out within a very short time if they are wetted. In Brazil these dried eggs are killed in gutters by means of a blow-flame, but in Georgetown the risk of fire was too great for this. The water supply of Georgetown and other places depends very largely on roof collection by gutters, and storage in barrels and drums. The latter are also a source of breeding, and one of the most difficult tasks has been to enforce mosquito-proofing or sealing of such containers.

Insecticides have been used, especially DDT, in houses, trains, aircraft, and motor-cars, with useful results. One point brought out is that when DDT is (wrongly) used to spray the air of a room, as is the custom with pyrethrum products, the result is not so satisfactory as when it is sprayed on the walls. Records are given of various experiments with DDT. Phenothiazine is useful for water containers—it kills insects in dilutions approaching those of chlorine in drinking water, and is harmless [in those dilutions] to higher animals.

Aircraft are inspected and sprayed ; in quite a large proportion living insects have been found and this is causing concern to public health, agricultural and veterinary officials alike, though no living *Aëdes aegypti* or anophelines were recorded during 1944 and the first half of 1945. Vaccination against yellow fever is available free of charge, and any person arriving at the coast from the interior is kept under surveillance for six days.

In 1945 there were 207 persons employed in the Yellow Fever Service, including probationer inspectors. The financing of the scheme is explained : from September 1939 to October 1, 1945, 359,688 dollars had been expended, mostly from Government and Colonial Development and Welfare Funds, but with a contribution from the Rockefeller Foundation. Tribute is paid to the Foundation for the scientific assistance it has given.

Plans for the future are set out and there is an interesting Appendix on yellow fever in British Guiana. *Charles Wilcocks.*

DENGUE AND ALLIED FEVERS.

HITTI, J. K. & KHAIRALLAH, A. A. **A Report on the Recent Epidemic of Dengue in Beirut, Lebanon, and some of its Complications.** *J. Palestine Arab Med. Ass.* 1946, July, v. 1, No. 5, 150–53.

An exceptionally severe epidemic of dengue occurred in Beirut in 1945, when the lateness of the rains and of the advent of the cold weather provided conditions in which the disappearance of the disease was delayed.

Early in the course of the epidemic a tendency to haemorrhage was observed in several patients ; petechiae were often seen. Among the other complications were : corneal ulceration, meningismus (often), post-febrile neuralgia (several cases), otitis media (fairly common), and acute gastritis (many cases). Including deaths from complications, the fatality rate was about one per cent.

John W. D. Megaw.

PLAGUE.

DEVIGNAT, R. Aspects de l'épidémiologie de la peste au Lac Albert. [**Epidemiology of Plague at Lake Albert.**] *Ann. Soc. Belge de Méd. Trop.* 1946, June 30, v. 26, Nos. 1/2, 13–54, 4 figs. [21 refs.]

This publication is an elaboration and application of a theory and scheme already propounded [see this *Bulletin* 1946, v. 43, 647], which gave a linear

equation, $Q=K\ [k(i+t)+k'(i'+t')+k''(i''+t'')\ \ldots]$ for the determination of the common indices, Q_1, Q_2, . . . Q_6, of epidemiogenic and epizootic transmissibility of plague. The author himself warns his readers that his is an attempt to systematize epidemiology, and that the data obtained will be of the order only of first approximations. Practical applications of the method are given in the second chapter; and in the third it is shown how the three fundamental planes of plague activity have been studied. Without attaching exaggerated exactitude to the figures obtained, it may be concluded that, as regards Lake Albert, the conditions there of transmissibility were favourable both on the sylvatic and the domestic plane and that there was no difficulty in passing from the one plane to the other. The persistence, or endemicity, of plague was thus easy to explain in spite of the long "silences" that can occur on the human plane. These intervals of freedom from plague on the human plane, or of difficult passage to it from the other two planes, are exhibited quantitatively by a fifty-fold decrease of the Q index. Moreover, with the arrival of plague on the human plane, it is even more difficult for plague transmitted by fleas to pass from man to man, since the local variety of human flea, *Ctenocephalides felis strongylus* is stated not to be a plague vector. Pneumonic plague is expressly excluded from these epidemiological investigations.

W. F. Harvey.

GUPTA, J. C., PANJA, G. & CHATTERJEE, M. **The Effect of Sulphadiazine and Penicillin on Experimental Animal Plague.** *Indian Med. Gaz.* 1946, June–July, v. 81, Nos. 6/7, 234–5.

Penicillin was found of no value.

BACILLARY DYSENTERY.

MACGREGOR, I. **Acute Enteritis in Subtropical Climates.** *Brit. Med. J.* 1946, Aug. 17, 225–6, 1 graph.

Observation of 455 cases of upper respiratory infection (acute nasal catarrh and acute sinusitis) and 357 cases of acute enteritis (simple diarrhoea not of specific dysenteric origin) over a period of 80 weeks suggested, by the close correlation of the weekly incidence of the two conditions, that the cause of acute enteritis might be related to the causative organism of upper respiratory infections.

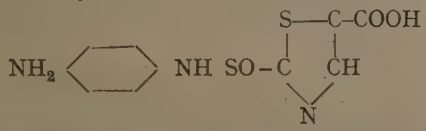
Sandstorms and *khamsins* [the strong wind of Egypt], which produced a marked increase in the incidence of acute exacerbations of chronic rhinitis and sinusitis, appeared to cause no increase in the incidence of fresh upper respiratory infections or in that of acute enteritis, suggesting thereby that mere mechanical irritation of the bowel does not suffice to explain the enteritis. There also appeared to be no connexion between the incidence of acute enteritis and any one specific ration issue. Sudden cold spells of weather or a sudden fall in the temperature at night, however, increased not only the incidence of acute respiratory infections but also that of acute enteritis.

Following the view that acute gastro-enteritis of children is probably due to virus invasion of the intestinal tract rendering the mucous membrane more vulnerable to the ordinary bacterial flora of the region, the author suggests that a similar mechanism might account for the acute enteritis of the subtropics. The absence of an associated gastritis could be explained on the ground that the virus, lodging first in the nasopharynx, reaches the intestine by invasion of the blood stream; that it passes directly from the nasopharynx to the intestine

through the oesophagus and stomach but that the higher resistance of the adult gastric mucosa prevents the development of gastritis ; or that in the adult stomach, possibly owing to its high *p*H, there are not the organisms to attack the mucosa when it has been damaged. *F. Murgatroyd.*

WINNEK, P. S. **An Intestinal Antiseptic: 2-Sulfanilamido-5-Carboxythiazole.** *Science.* 1946, June 21, 719–20.

2-sulphanilamido-5-carboxythiazole has the formula :—



It is a white crystalline solid, slightly soluble in water (40 mgm. in 100 cc. at 24°C.) giving an acid solution. The solubilities of the mono and disodium salts are greater than 30 per cent., the *p*H of saturated solutions being 5·4 and 8·5 respectively. The solubility of sodium salt of the acetyl derivative is 8 per cent., so that there is little danger of the kidney being blocked by a deposit of crystals. When tested *in vitro* against bacteria such as streptococcus, pneumococcus, staphylococcus, and enteric organisms, its bacteriostatic activity was usually greater than that of sulphanilamide, and equal, or slightly inferior, to that of sulphathiazole and sulphadiazine.

When given in single doses to mice the LD_{50} is 8·0 gm. per kgm. by mouth or 5·0-6·0 gm. per kgm. by intraperitoneal administration. The chronic toxicity in mice, rabbits and dogs was much less than that of the readily absorbed sulphonamides and was comparable to that of succinyl sulphathiazole. When given to man (0·25 gm. per kgm. per day for 5 days) the blood concentration is always low (less than 1 mgm. per 100 cc.) ; about 3–11 per cent. of the amount taken by mouth is excreted in the urine. In 4 men taking this dose, the number of coliform bacilli in the faeces was reduced to 10–1,000 organisms per gm. of wet faeces in 24–48 hours ; it is claimed that the new drug is more effective than succinyl sulphathiazole in this respect. HARRIS and FINLAND (*Proc. Soc. Exper. Biol. & Med.*, 1945, v. 58, 116) have tried it in cases of bacillary dysentery ; they found it to be absorbed into the blood only in small amounts, to be non-toxic, and to be effective in treating the dysentery. *F. Hawking.*

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS.

LEITMAN, M. Z. [**The Virulence of various Strains of the Dysentery Amoeba and the Effect of Bile on the Infestation by Protozoa.**] *Med. Parasit. & Parasitic Dis.* Moscow. 1945 [1946], v. 14, No. 6, 39–44.] [In Russian.]

With a view to throwing some light on the epidemiological rôle of symptomless carriers of *Entamoeba histolytica*, the author carried out a series of experiments to test the virulence of various strains for kittens and pups. The following four strains were used :—(1) from a symptomless carrier ; (2) from a chronic case during the latent period ; (3 and 4) from cases of acute amoebic dysentery.

The animals (total number 46) were inoculated either *per os*—with cysts, or *per rectum*—with the active amoebae. Half of the animals were inoculated without any preliminary treatment, whereas the other half received a dose of bile *per rectum* (2 cc. in kittens, 3 cc. in pups) preceded by an enema. In the

first group only 11 out of 23 (48 per cent.) acquired an infection, and there was no essential difference in the incubation period, course of infection and intestinal lesions produced by the four strains. However, in the second group (with bile) the results were more striking, the number of infected animals being 16 (70 per cent.). The effect of bile treatment upon infectivity was especially marked in those strains which proved to be less virulent when inoculated without bile.

The effect of bile upon the infectivity of protozoa was also tested by inoculating white rats with a strain of *Balantidium coli* isolated from a symptomless human carrier. Whereas among 17 rats inoculated without bile only one (5·9 per cent.) became infected; in a group of 15 rats treated with bile 5 (33·3 per cent.) acquired an infection.

Bile irritates the mucous membrane of the intestine, producing hyperaemia, abundant secretion of mucus and desquamation of the epithelium. These changes apparently sensitize the intestinal wall, rendering the experimental animals more susceptible to infection with intestinal protozoa.

It is concluded that the results of these experiments have again demonstrated that strains of *E. histolytica* isolated from symptomless carriers are potentially not less virulent than those originating from patients suffering from dysentery.

[While this conclusion may be right, it should be emphasized that, like previous experiments of this kind, it is based on and applies to the behaviour of *E. histolytica* in non-natural hosts.] *C. A. Hoare.*

LINDSKOG, G. E. & WALTERS, W. **Surgical Aspects of Amebic Dysentery.** *J. Amer. Med. Ass.* 1946, May 11, v. 131, No. 2, 92–5, 3 figs.

This is a report of three cases of amoebic hepatic abscess, and one of amoebic granuloma of the caecum, occurring among personnel of the U.S. Army and Navy returning after service in the tropics. In one of the cases the abscess perforated through the diaphragm to the pleuro-pulmonary region, and in another a right hepatic abscess recurred after open drainage, but subsided after a course of penicillin and emetine, without further operation. The indications for open drainage in preference to aspiration are discussed. The amoeboma occurred in a patient who had suffered from acute amoebic dysentery twelve months previously, for which he had been treated, apparently successfully, with emetine and carbarsone; he relapsed six months later, and after further medical treatment with only slight improvement, a moveable tumour appeared in the right lower quadrant, and X-ray studies revealed a constrictive filling defect at the ileo-caecal junction. Right hemicolectomy was performed and was followed by uneventful recovery. In none of these four cases were amoebae or cysts demonstrated in the stools. *W. L. Harnett.*

i. SMYTH, M. J. **Confusion of Amoeboma with Carcinoma.** *Lancet.* 1946, Sept. 14, 376–8, 1 fig.

ii. HAWE, P. **Amoeboma and Carcinoma.** [Correspondence.] *Ibid.* 1946, Oct. 5, 508–9.

i. Among the many abdominal cases admitted to the Ministry of Pensions Hospital at Roehampton, London, there were some in which left iliac colostomy had been performed for no ascertainable reason. A factor common to all was that they all had suffered from amoebic dysentery and all, except one, had been treated by emetine injections. The logical conclusion was that an amoeboma had been mistaken for carcinoma. Radical surgery in such cases, such as resection of the colon or excision of the rectum, may be serious because in the absence of anti-amoebic treatment, a fatal outcome is almost invariable.

Occasionally, as in the case recorded by MORGAN [see this *Bulletin*, 1945, v. 42, 124] amoeboma and carcinoma may coexist.

Amoebiasis of skin and subcutaneous tissues may develop after colostomy, as in the case cited in this paper. This patient was admitted with the diagnosis of rectal carcinoma. He had served in many countries, but had never had "dysentery". As the result, he claimed, of lifting heavy shells, a lump appeared in the left lower abdomen, an occurrence associated with diarrhoea tenesmus and piles. On admission to hospital, no amoebae having been demonstrated in the faeces, a left iliac colostomy was performed. Subsequently a large infected area appeared in the vicinity of the bowel and further spread was prevented by diathermy excision.

After closure of the colostomy, the wound became acutely inflamed and broke down, with parting of the sutures in the bowel and re-establishment of the colostomy. During the next four weeks the infected area continued to spread and the patient's condition was rapidly deteriorating, when *E. histolytica* was demonstrated in the faeces. On intramuscular emetine treatment reinforced by diodoquin (4 tablets daily for ten days) the stools became clear of amoebae and the wound rapidly scarred over.

Two cases of the converse error are recorded in which carcinoma of the rectum had been treated for dysentery; in both, rectal digital examination had been omitted.

ii. This correspondent refers to the rare existence of amoeboma of the rectum, which may be present as an ulcer or as a papilliferous overgrowth, and which may be indistinguishable from carcinoma.

A course of emetine, before resort is had to operation, is a valuable aid to diagnosis, but only a complete resolution of the amoeboma can be accepted as proof of a purely amoebic origin, by this means.

The author has seen six of these cases during four years military service. Five resolved on medical treatment. The sixth was diagnosed as an advanced carcinoma of the rectum; colostomy was proposed, but a proctoscopic examination on the operation table revealed the presence of *E. histolytica*. Operation was postponed and the patient died despite a few days of emetine treatment. Post-mortem examination showed how futile a colostomy would have proved, for the whole colon was diffiusely ulcerated.

Whilst colostomy may be required where other methods fail, a note of warning is sounded regarding too hasty embarkation on this procedure, owing to the risk of spreading amoebic infection and to serious hepatic complications.

P. Manson-Bahr.

CROPPER, C. F. J. **Amoeboma of the Hepatic Flexure.** [Memoranda.] *Brit. Med. J.* 1946, June 29, 988.

The case reported is that of a Gurkha, aged 23, with no previous history of ill health. For three weeks he had complained of pain in the right hypochondrium and of evening pyrexia, unaccompanied by bowel symptoms. In the right hypochondrium was a rounded, hard, tender swelling, 3½″ in diameter, which appeared to be unconnected with the liver, and was attached to the right rectus. The stools were repeatedly negative for exudate, cysts and ova; the white cell count was 14,000 per cmm. with 80 per cent. polymorphs; and sigmoidoscopy showed typical amoebic ulceration, but no scrapings were taken. Skiagrams after a barium enema showed arrest of the barium by a constriction at the hepatic flexure, with delayed filling of the ascending colon and caecum, and retention of the opaque medium in that part of the intestine after evacuation of the enema. A course of emetine and penicillin led to rapid resolution of the swelling and a skiagram three weeks later showed a normal large intestine;

on sigmoidoscopy all the ulcers were found to be healed. Throughout the illness there were never any dysenteric symptoms. *W. L. Harnett.*

HALAWANI, A., NOR-EL-DIN, G. & AWNI, A. Y. **Treatment of Amoebic Dysentery with Sulphaguanidine as compared with Emetine and other Antidysenteric Drugs.** *J. Roy. Egyptian Med. Ass.* 1946, Jan.–Feb., v. 29, Nos. 1/2, 13–19.

"(1) Sulfaguanidine exerts a favourable effect on the clinical manifestations of amoebic dysentery, but relapses occur shortly after cessation of treatment.

"(2) The average time after which relapses occur after sulfaguanidine is approximately ten days, while this time after emetine treatment is as long as 21 to 27 days.

"(3) The action of sulfaguanidine is solely on the vegetative forms. The cysts are not affected by this drug. In some cases under sulfaguanidine treatment the amoebae changed from the vegetative forms to the cystic stages.

"(4) Resistant cases have been found to benefit from a combined treatment of emetine and sulfaguanidine.

"(5) The effect of enterovioform and dysenteran on the vegetative forms of *E. histolytica* was found inferior to emetine. Their effect was mainly on the cysts."

OSTROUMOV, V. G. [**On the Protozoal Fauna of the Intestine in Post-Dysenteric Colitis (Dysenteria Chronica).**] *Med. Parasit. & Parasitic Dis.* Moscow. 1945 [1946], v. 14, No. 6, 44–6. [In Russian.]

The author describes the results of a survey in Stalinabad (Middle Asia) of the protozoal fauna of hospital patients (total 115) suffering from chronic (post-dysenteric) colitis. The examination was carried out mainly in fresh and iodine-treated faecal preparations, and sometimes in fixed and stained material. When the results were negative, the faeces were examined repeatedly (up to 6 times, and on the average 2·6 times). Only 49·6 per cent. of the patients examined were infected with intestinal protozoa, in most cases with one or two species (24·3 and 17·4 per cent. respectively). The relative incidence of the various parasites was as follows: *Entamoeba coli* 20·9 per cent.; *Chilomastix* 20 per cent.; *Trichomonas* 18·3 per cent.; *Giardia* 12·2 per cent.; *Iodamoeba* 7·8 per cent.; *Endolimax* 4·3 per cent.; *Entamoeba histolytica* 2·6 per cent.

In the course of repeated examinations the frequency of the occurrence of different parasites in the same person varied, the findings in 297 examinations of 115 patients being distributed as follows: *Chilomastix* 32 times; *E. coli* 29 times; *Trichomonas* 25 times; *Giardia* 23 times; *Iodamoeba* 9 times; *Endolimax* 5 times; *E. histolytica* 3 times. The author compares his findings with those in 743 healthy adults, examined during the same period by another worker, whose results were as follows: *E. coli* 37·9 per cent.; *Chilomastix* 15·8 per cent.; *Trichomonas* 16·1 per cent.; *Giardia* 17·4 per cent.; *Iodamoeba* 22·1 per cent.; *Endolimax* 7·9 per cent.; *E. histolytica* 0·4 per cent.; *Balantidium* 0·5 per cent. This comparison showed that in cases of colitis the finding of *E. coli*, *Iodamoeba* and *Endolimax* is reduced to half, while *Chilomastix* and *Trichomonas* are almost as common in cases of colitis as in healthy intestines. This confirms the results obtained by other Russian workers, who found that there was a marked diminution of the protozoal fauna when the normal functions of the bowel were disturbed. *C. A. Hoare.*

LEITMAN, M. Z. & VITLINSKAYA, I. A. [**Treatment of Carriers of Pathogenic Protozoa.**] *Med. Parasit. & Parasitic Dis.* Moscow. 1945 [1946], v. 14, No. 6, 46–50. [In Russian.]

In view of the epidemiological rôle of carriers of *Entamoeba histolytica* in the transmission of the infection and of the possible transition from symptomless infection to clinical forms of the disease, the authors have carried out an experiment on the treatment of "cyst-carriers" of *E. histolytica* in Tashkent. A preliminary examination of 1,002 persons revealed the following incidence of pathogenic intestinal protozoa: *E. histolytica* 127 (12·7 per cent.), *Giardia* 139 (13·9 per cent.), *Isospora* 7 (0·7 per cent.), *Balantidium* 1 (0·1 per cent.). The infected persons were dealt with as follows: one group of *E. histolytica* carriers was treated with yatren (adults had 4 courses of 0·5 gm. administered twice a day for 5 days with intervals of 5 days between the courses; children had 4 courses of 0·25 gm. a day for 6 days with intervals of 6 days); a second group of *E. histolytica* carriers was treated with osarsol (for adults 5 courses of 0·25 gm. thrice a day for 5 days with intervals of 5 days; children had 5 courses: dosage as for yatren); a third group, comprising *Giardia* carriers was treated with acriquine [=mepacrine] (adults received 0·1 gm. thrice daily for 5 days, followed by a 10-days interval, after which the same dose was repeated for 3 days, again followed by an interval of 10 days and a repetition of treatment for 3 days; children were given 0·1 gm. daily for one month).

Throughout the course of treatment the stools of the carriers were examined once a week, and subsequently once a month for five months. During the first period of observation *E. histolytica* cysts were still present in 20·4 per cent. of carriers treated with yatren and in 14·4 per cent. of those treated with osarsol, but the percentage of positive results fell considerably in the course of the first three months. However, from the fourth month it began to rise again, especially in the yatren group, reaching 26·6 per cent. after five months, *i.e.* it was higher than during the period of treatment; but in the osarsol group an increase of positive findings was noted only after five months. In the group of *Giardia* carriers treated with acriquine there was also at first a diminution in the number of cyst-passers, followed by an increase after three months, reaching 27·7 per cent. at the end of the fourth month and 27·5 per cent. after the fifth. On the whole, in the majority of carriers the treatment was effective for only 3–4 months, after which cysts again appeared in over 33 per cent. of the cases. It is concluded that carriers among food-handlers can be dealt with effectively only if treated at least twice a year. *C. A. Hoare.*

ACOSTA MATIENZO, Josefina. **Incidence of Intestinal Protozoa among Patients of the University Hospital at San Juan, Puerto Rico.** *Puerto Rico J. Pub. Health & Trop. Med.* 1946, June, v. 21, No. 4, 369–71. [Spanish version 372–5.]

HALAWANI, A. & EL KORDY, M. I. **A Case of Human Coccidiosis.** *J. Roy. Egyptian Med. Ass.* 1946, Jan.–Feb., v. 29, Nos. 1/2, 32.

MALDONADO, J. F. *Isospora hominis* **Fantham, 1917, in Puerto Rico. A Report of the First Case observed.** *Puerto Rico J. Pub. Health & Trop. Med.* 1946, June, v. 21, No. 4, 397–400, 2 figs. [Spanish version 401–4.]

"*Isospora hominis* occurs in Puerto Rico, as demonstrated by the discovery of a case in which the chances of infection were entirely local. The presence of *S. mansoni* obscured the possible effects of the coccidium on the patient, but there is the possibility that his recent intestinal disorders may be ascribed to

this parasite. The course and general character of the infection were similar to those described by most authors, but the action of fuadin on the coccidiosis was questionable."

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

RAFYI, A. Sur la fièvre récurrente sporadique en Iran. Etude expérimentale de *Spirochaeta persica* (deuxième note). [**On Sporadic Relapsing Fever in Iran. An Experimental Study of** *Spirochaeta persica* **(2nd Note).**] *Arch. Inst. d'Hessarek.* Teheran. 1946, Jan., v. 2, No. 2, 37–42.

The author, in collaboration with DELPY [see this *Bulletin*, 1939, v. 36, 757], recorded the existence of human relapsing fever in the north and north-west of Iran. They also showed that the causal agent was *Spirochaeta persica* and that it was transmitted by all stages of *Ornithodorus tholozani*.

Subsequently the disease has been found in other parts of Iran and especially in Teheran, and 16 cases have been observed at the Hessarek Institute in the last five years. As a rule the patients recovered spontaneously and treatment with novarsenobenzol was efficacious.

The author has studied the infection in guineapigs, rabbits, rats, and mice, all of which may be infected, not only by the injection of blood containing spirochaetes, but also by the bites of infected ticks. One in five of ticks collected in infected houses produced infection when fed on normal rats. The infection was transmitted hereditarily to the offspring of infected female ticks. Adults were found to be still infective after 1,175 days, and nymphs infected in this stage were stated to be still infective after 1,403 days. Ticks emerging from eggs laid by infected parents were still infective 2,066 days after the parent was infected.

The spirochaete was found to survive in the brain of white rats up to 593 days. In a footnote the author corrects an error in their earlier paper and states that residual infections in the brain of rabbits, guineapigs, dogs and sheep have *not* yet been demonstrated. *E. Hindle.*

ANDERSON, E. S. **Penicillin in the Treatment of Experimental Relapsing Fever in Rats.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1946, Aug., v. 40, No. 1, 93–100.

This is a record of experiments carried out to test the effect of penicillin in white rats inoculated with a Cyprus strain of *Spirochaeta recurrentis*, which in nature is transmitted by *Ornithodorus tholozani*, and is apparently insensitive to arsenicals.

One group of rats was given massive doses of penicillin either at the time of inoculation of spirochaetes, or three hours later, or at both these times. This treatment was quite ineffective in preventing infection, but an intermittent prophylactic dose maintained three-hourly for 78 hours seemed to prevent the development of the disease.

Once spirochaetes had appeared in the circulation, intermittent dosage did not control the disease, and the persistence of the infection in the brain of a rat was not influenced by relatively huge doses of penicillin administered early in the disease.

The *in vitro* exposure of spirochaetes to high concentrations of penicillin did not affect their structure, motility, or infectivity.

Professor S. ADLER and Dr. R. ASHBEL [personal communication to the author], using a similar strain of spirochaete, have carried out a comparable

set of experiments, also with negative results. It would seem, therefore, that the Cyprus strain of *S. recurrentis* is not only resistant to arsenicals, but also to penicillin. *E. Hindle.*

RANDOLPH, N. M. **DDT to control the Relapsing Fever Tick.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 396.

"The results as given in table I show that DDT sprays of 10 per cent. and 20 per cent. strengths were effective in killing relapsing fever ticks. Applying DDT spray directly to the tick does not appear to increase the effect of the insecticide. DDT of high concentrations is a promising insecticide for controlling these ticks, however, the cost and feasibility of applying it to natural conditions may be prohibitive. Field experiments are being conducted."

DAS GUPTA, B. M. & SEN, R. L. **Canine Leptospirosis in Calcutta.** *Indian J. Vet. Sci. & Animal Husbandry.* 1945, Mar., v. 15, Pt. 1, 52–4, 2 figs. on 1 pl.

"A case of leptospiral jaundice in the dog caused by *Leptospira icterohaemorrhagiae* is described."

YAWS.

HALAWANI, A. & NOR-EL-DIN, G. **A Case of Juxta-Articular Nodes in a Syphilitic Patient.** *J. Roy. Egyptian Med. Ass.* 1946, Jan.–Feb., v. 29, Nos. 1/2, 80–83, 1 fig.

SOMPAYRAC, L. & SPENCE, H. **A Yaws Clinic in New Georgia.** *Arch. Dermat. & Syph.* 1946, Aug., v. 54, No. 2, 205–6.

GUIMARÃES, F. N. Ensaios terapêuticos com penicilina. V. Bouba (Framboesia, pian, yaws). I. Dose curativa minima. II. Redução do número de injeções diarias. III. Redução de tempo de tratamento pelo emprêgo de doses mais elevadas. [**Penicillin Therapy. V. Yaws.**] *Mem. Inst. Oswaldo Cruz.* 1945, Aug., v. 43, No. 1, 31–58, 15 figs. [Summary in English by the author.]

"This report belongs to the series of works carried out at Oswaldo Cruz Institute, on the treatment of treponematosis with penicillin.

"The present report deals with investigations performed in order to ascertain the following points :—(1) the minimal curative doses for the initial lesions of yaws ; (2) the effect of reduction of the number of injections each day, to verify the possibility of application of penicillin in the prophylaxis of yaws in rural zones ; (3) reduction of the time of treatment by application of high doses.

"(1) With doses of 150 and 100 Oxford units every four hours, clinical recovery was obtained after 17 days of treatment. With 50 O.u. during 40 days clinical recovery was not obtained.

"(2) (*a*) With 3 injections of 400 O.u. each day (at 6, 12 and 18 hours), clinical recovery was obtained after 14 to 16 days ;

(*b*) with 2 injections of 400 O.u. each day (at 6 and 18 hours), clinical recovery was obtained after 16 to 23 days ;

(*c*) with 1 injection of 1,600 and 3,200 each day, clinical recovery was obtained after 30 and 20 days.

" (3) With doses of 33·3 and 46·7 O.u. for each kilo of weight every four hours, during 15 days, clinical recovery was obtained more or less in 25 days. The same result was obtained with the doses of 61·5 and 166·7 O.u. for each kilo of weight, every four hours, during 4 days. But with 100,000 O.u. in five doses of 20,000 a day, clinical recovery was not obtained."

LEPROSY.

COCHRANE, R. G. **The Classification of Leprosy.** *Leprosy Review.* 1946, July, v. 17, No. 2, 36–42.

The author gives his views on this admittedly difficult subject in the present paper. The Cairo Conference classification of 1938 was to have been reviewed at a succeeding one, which it has not yet been possible to hold. In the meantime the matter has been much discussed by South American workers, who have set out their views at length in the *Revista Brasileira de Leprologia* [this *Bulletin*, 1946, v. 43, 451] based on clinical, structural and immunobiological factors. The neural and lepromatous forms are regarded as " polar " forms, between which there is an adequately charted no-man's land. The uncharacteristic cases are divided by Pardo-Castello into :—(*a*) non-specific inflammatory tissue reactions, and (*b*) borderline cases between tuberculoid and lepromatous cases, which are transitional and may change one into another. The present author, however, does not believe that tuberculoid cases can change into lepromatous ; and he discusses in detail the histological changes in the different forms on the usually accepted lines. Those interested should refer to the original paper for details. *L. Rogers.*

RADNA, R. Sur les formes de la lèpre dans le district de l'Uele. [**Leprosy in Uele.**] *Ann. Soc. Belge de Méd. Trop.* 1945, Dec. 31, v. 25, Nos. 3/4, 205–17.

The Uele District of the Belgian Congo is situated in the north-eastern area of that country. Leprosy is reported to have increased there in recent times as the result of invasion by Arab slave dealers. The indigenous people are well able to recognize the disease ; they treat cases ineffectually by vegetable caustics. The large proportion of early cases indicates that the disease is still on the increase. Tables of the percentages of different forms of leprosy show 30·8 per mille of neural and 3·2 per mille of lepromatous cases, making a total of 34 per mille. Bacteriological examinations of nasal scrapings, gland juice and biopsy material were made in 7,600 patients, with the result that nerve cases rarely showed more than 1 to 5 bacilli per field of the microscope, but all the typical lepromatous lesions showed large numbers of bacilli. Inquiries showed a history of 43·5 cases per mille in adults of the present generation, 8·3 per mille in their parents and 6·4 per mille in the grandparents : this supports the view that a rapid increase of the disease has occurred in recent times. Attempts have been made to carry out voluntary segregation in agricultural villages of a self-supporting character, but with only a moderate degree of success. The author therefore advocates concentrating the work in the most severely infected areas, with the segregation of infective cases under medical supervision, and separate accommodation for the slightly infective, but mutilated, nerve cases. Patients with active disease, especially children, should be treated at dispensaries. Propaganda in support of the segregation and treatment of infective patients, together with the examination and control of contacts of infectious patients, are also required. *L. Rogers.*

FIELDING, J. W. **Observations on Human Leprosy : Infection of Rats with Human Excretal Organisms.** *Med. J. Australia.* 1946, Apr. 27, v. 1, No. 17, 578–85. [24 refs.]

The author holds that the digestive and genito-urinary systems of leprous rats may be highly infected, and that the organisms from these sites play a major rôle in the dissemination of the disease in rats [see this *Bulletin*, 1945, v. 42, 903]. He therefore examined the urine and faeces of patients in the New South Wales Leprosarium, and found numerous acid-fast organisms, especially in the faeces. The Breed bacteria count method was used to estimate the numbers of such acid-fast bacilli in the dried faeces of leprosy patients : counts of from 75 to 500 millions per gramme of faeces were obtained. Such dried faeces were therefore used to infect rats by repeated inunction on scarified skin, in skin slits and by subcutaneous inoculation, with the result that acid-fast bacilli were subsequently found both locally and in small numbers internally in the rats. Details of experiments by the inoculation of haemolysed blood from leprosy patients into rats are recorded, and it is suggested that the method might be used for the diagnosis of leprosy. Cases of leprosy excreting acid-fast bacilli are considered to be dangerous carriers. Climate is considered to be of little importance, except in so far as it may affect the duration of the vitality of lepra bacilli of faeces in the soil. Rats also excrete numerous acid-fast bacilli and their dried excreta are said to remain infective up to two years.

L. Rogers.

ROW, R. **Isolation of *Mycobacterium leprae* in Culture.** *Indian Physician.* 1946, Apr., v. 5, No. 4, 75–81, 2 pls.

The author has sought for some method of cultivating *Mycobacterium leprae* in symbiosis with some other organism. The work of GREVAL, LOWE and BOSE and GREVAL, SEN GUPTA and NAPIER [see this *Bulletin*, 1939, v. 36, 537 ; *ibid.*, 1030] on the complement fixation test in leprosy (and also in kala azar) by the use of an antigen from cultures of tubercle bacilli led him to try adding a weak saline suspension of lepra bacilli obtained from a nodule to a tube of haemoglobinized saline. After it had remained free from extraneous contamination for four days, he added a drop of a ten-day culture of *Leishmania* and incubated at 20°C. At the end of a week marked proliferation of acid-fast bacilli was found in close relation to *Leishmania* rosettes. After another week subcultures of the bacilli were obtained on both solidified saline agar and on glycerinated potato. The former at 20°C. showed a symbiotic culture ; the latter at 37°C. showed only acid-fast bacilli. As positive results have been obtained in four successive cases he claims to have cultivated the true causative organism of leprosy. A number of illustrations of the cultures are included in the paper. The bacillary cultures were pleomorphic, and after three weeks they became coccobacillary. He next used the same method with success for the symbiotic culture of the *Mycobacterium* of rat leprosy and he hopes before long to be able to report the results of inoculating rats and mice with such cultures. Confirmation of these results will be awaited with interest. *L. Rogers.*

COELHO, J. T. Nodulos hipodérmicos, pseudo-sarcóidicos, de etiologia leprosa. [**Pseudo-sarcoids ; Subcutaneous Nodules.**] *Rev. Brasileira Leprologia.* S. Paulo. 1945, Dec., v. 13, No. 4, 287–92, 4 figs.

Detailed descriptions are given of patients presenting subcutaneous nodules in various situations—wrist, dorsum of the hands, forearms, elbows, the legs and the cheeks. Puncture fluid revealed *Myco. leprae* and sections of material

obtained by biopsy showed the usual leprotic tissue with the organisms. When these tumours are numerous, little can be done beyond the giving of chaulmoogra ; but if they are few, good results may be obtained by removal of them.

H. Harold Scott.

GRUENFELDER, B. & LASCH, W. **Leprosy of the Nose in relation to the Sexual Apparatus.** *Acta Med. Orientalia* (*Palestine & Near East Med. J.*). 1946, May, v. 5, No. 5, 156–61. [43 refs.]

The authors have previously found, in cases of infantile cryptorchidism, anomalies in the olfactory sphere. In the present paper they discuss a similar relationship in cases of leprosy and they quote from a number of writers in support of this view. They have noted, in male lepers, effeminate features usually only met with in eunuchoids ; and in nodular and mixed cases, remarkably early reduction of sexual functions is to be observed. Those who are attacked before the age of puberty may show arrest in the development of the testis and of secondary sex characters, with, according to GLUECK, absolute impotence; or loss of sexual powers in 95·4 per cent. of nodular cases. The sexual apparatus of women in nodular cases is reported to be affected in the same way as in men, with resulting dysmenorrhoea and sterility ; and in females attacked young, there is absence of menstruation later on. It is in just these nodular cases that the nasal mucous membrane is also affected in leprosy, as shown by references to papers of older writers. The analogous behaviour of the sexual system and the nose indicates that something in common connects them, but further investigations are required to enable the precise relationship to be determined.

L. Rogers.

PORTUGAL, H. & ROCHA, G. L. **Juxta-Articular Node of Leprous Origin.** *Arch. Dermat. & Syph.* 1946, May, v. 53, No. 5, 471–6, 5 figs. [Refs. in footnotes.]

This condition is most commonly seen in syphilis and yaws ; it has also occasionally been observed in tuberculosis. The author now records a case of leprosy with a nodule the size of a chestnut near the upper epiphysis of the right ulna, together with other leprous nodules in various parts of the body. On excision and microscopical examination, as shown by illustrations, it was found to be fibrous in nature with numerous capillaries and small cellular infiltrates. The central zone showed numerous lepra cells of Virchow with many acid-fast bacilli typical of a leproma. *L. Rogers.*

HARLEY, R. D. **Ocular Leprosy in Panama. A Study of 150 Cases.** *Amer. J. Ophthalm.* 1946, Mar., v. 29, No. 3, 295–316, 19 figs. [37 refs.]

One hundred and fifty cases of ocular leprosy are reviewed by Harley. Twenty-six per cent. were classified as belonging to the anaesthetic form and seventy-four were mixed, *i.e.* a combination of the nodular and anaesthetic types. Nearly all the mixed cases showed a positive blood test for syphilis, whilst the neural cases were less likely to give a positive test unless there was an acute tuberculoid reaction. It is generally agreed that lesions of the eye present a very real and serious complication in leprosy, and ocular involvement varies from 20 to 100 per cent. of all patients when the different forms of the disease are taken into account. In Panama 90 per cent. of the leprous patients were found to have ocular involvement, not including the adnexa. If the adnexa were included, it may be assumed that in predominantly nodular leprosy all but those in the first few years of their disease had involvement about the

eyes. Some form of ocular leprosy will eventually occur if the patient lives long enough. The 10 per cent. of eyes free from the disease were found exclusively in recent cases. Among all the organs involved in leprosy, the ocular lesions appeared to cause more complaints than any others.

Not only is the method of transmission of leprosy in dispute, but there is still much speculation as to the medium of transfer. The portal of entry for the ocular invasion is also in question. By some it is believed that the bacilli enter the eye externally from the conjunctiva or episcleral tissues. Most workers believe that the ocular infection is secondary to a haematogenous spread. Fuchs found the primary ocular lesions in the anterior part of the ciliary body and major circle of the iris, and is of opinion that the bacilli may appear in healthy tissues, but do not necessarily affect them. In the case of five sectioned eyes it was shown that inflammation occurred in the perilimbal episclera and the longitudinal fibres of the ciliary muscle, extending back to the ora serrata, but no further. In all cases the cyclitis appeared to be an older inflammation than the episcleritis, which added to the evidence of an endogenous spread. There appear to be two factors, exogenous and endogenous, which seemed to be acting simultaneously in the disease. In some eyes, one factor often predominates over the other. The disease primarily strikes the anterior segment of the eye, and it is in this region that bacilli are most often encountered. It is unusual to see any pronounced ocular complication not accompanied by varying degrees of nasal mucous-membrane ulceration and perforation. The possibility of an ascending infection by way of the naso-lachrymal duct is to be considered, but purulent dacryocystitis is not seen as commonly as might be suspected.

In the series of 150 patients, 13 per cent. were found to be totally blind in one or both eyes, 41 per cent. had vision of 6/60 or less in one or both eyes, 43 per cent. had normal vision in both eyes, 10 per cent. had normal vision in one eye. Fifty-four per cent., therefore, may be regarded as the percentage with severe ocular complications of leprosy. The adnexa of the eye, the region of the eyebrows and eyelids, are involved with considerable frequency. Fifty per cent. of the patients had hypertrophy of the skin of the brows, or cutaneous nodules, with varying degrees of loss of hair of the eyebrows or lashes (madarosis). In nodular leprosy, formation of nodules about the outer eyebrows is common. The margin of the eyelid, usually the upper, may be attacked by nodules and these nodules may ulcerate ; a diffuse infiltration of the eyelid margins may occur, which gives the edges a rolled, thickened appearance (tylosis).

In anaesthetic leprosy, isolated areas of diminished or absent sensitivity occur in the skin about the eyes. These may differ slightly in colour from the rest of the skin. In 10 per cent. of cases there was paresis of the orbicularis palpebrarum (lagophthalmos) to the extent that there was incomplete closure of the lids. Fully another 10 per cent. of patients showed weakness of the lid closure. In each case the involvement was bilateral, but differed in intensity between the right and left side. The cases of lagophthalmos were in the purely neural and mixed types alike. The affection appeared to be a pure neural one and involved the eyelids out of proportion to the other structures. Ectropion resulting from lagophthalmos was found in most of these cases. Exposure keratitis and excessive lachrymation are also the usual sequelae.

Chronic conjunctivitis occurred in 40 per cent. of the cases and was of the catarrhal type, with profuse mucopurulent discharge. This caused less inconvenience than one would expect, probably because of the diminished sensitivity of the conjunctiva and cornea. Complete absence of sensation of the conjunctiva was, however, never demonstrated. Nodules on the conjunctiva were found in only two of the 150 cases, and the conjunctiva is apparently more immune than the other ocular structures.

The cornea is the most vulnerable of the ocular tissues and was involved in 87 cases; frequently it was the only site in which any pathological change could be seen. The most commonly observed corneal lesion was superficial keratitis, which occurred in over 34 per cent. of all cases. This usually began as a light milky haze punctuated by tiny white spots at the superior limbus. The lower margin was well delineated by a wavy line and was 2 to 3 mm. above the pupillary centre of the cornea, so that vision was not impaired. Pannus is usually present. In 23 cases an interstitial keratitis was present. In three cases the giant leproma occurred; this is the most unusual lesion of the cornea. It is engrafted on the cornea from a superficial keratitis and is teeming with lepra bacilli. "Beading" of the corneal nerves was found in five cases. Corneal anaesthesia was found to be rare.

Second only to the cornea, the iris is the structure most frequently involved in ocular leprosy. Iritis or its sequelae were recognized in over 50 per cent. of the cases. It was usually bilateral. Iris atrophy is common in the older cases. In old iritis the tiny lepromata were seen in 26 per cent. of the cases. Typically they were greyish-yellow, pedunculated, pin-point bodies about the pupillary border of the iris. The ciliary body and the anterior choroid were also probably involved, but this was more difficult to demonstrate clinically. Cyclitic membranes were found in advanced cases and leprous involvement of the ciliary body caused destruction of the zonule and subluxation of the lens. Secondary glaucoma is not common, contrary to what one might suspect. Leprosy of the sclera is nearly always secondary to limbal involvement. Lens opacities secondary to plastic iritis occurred in 20 per cent. of cases. Ophthalmoscopic examination of the fundi of 50 patients produced negative results, and proved that ocular leprosy is characteristically a disease of the anterior segment.

Prophylaxis for leprous eyes through protection with dark glasses is advised. Treatment is largely confined to palliative measures. Thyroxin solution (Squibb), used as drops, may be of aid in helping to clear corneal infiltration. The leprous patient withstands surgery of the eye well, despite the chronic inflammation. Surgery of the eyelids produces gratifying results.

E. O'G. Kirwan.

NEVES, A. Sarna crostosa. [**Scabies in Leprosy.**] *Rev. Brasileira Leprologia.* S. Paulo. 1945, Dec., v. 13, No. 4, 251–60, 11 figs. [15 refs.]

[This and the following three abstracts deal with papers read at the Leprosy Congress held at Minas Geraes in June 1945.] Two cases are recorded of the lepromatous type of leprosy associated with scabies infestation. The hands, forearms, buttocks, thighs and legs and, in one case, the left scapular region, were involved. Locally, there was incrustation and much itching, and the fingers showed deep fissures (clearly depicted in a photograph). The itch mite and also acid-fast bacteria were seen by microscopical examination. Association of scabies with the nervous type of leprosy has also been reported and the author states that the anaesthetic leprotic condition does not affect the infestation by scabies.

H. Harold Scott.

ALEIXO, Josefino & COELHO, J. T. Considerações sobre a reação de Montenegro em doentes de lepra. [**The Montenegro Reaction in Lepers.**] *Rev. Brasileira Leprologia.* S. Paulo. 1945, Dec., v. 13, No. 4, 263–6, 1 fig.

As regards the intradermal reaction of Montenegro, ARANTES has stated [see this *Bulletin*, 1942, v. 39, 170]: (1) That it is always negative in lepers with no history of leishmania infection; (2) that in lepers who had suffered years

before from leishmaniasis it was strongly positive; (3) that leprosy does not affect the reaction in patients cured of leishmaniasis; (4) that those with leishmania in an ulcer do not give the reaction.

The authors have not been able to confirm the last of these statements and quote the case of a leprous subject, aged 39 years, of the L_2N_1 type with generalized lepromata and anaesthesia of the forearms, hands and anterior surfaces of the knees, legs and feet. When the reaction was tried, a local erythematous papule formed in 24 hours, increasing in size and developing a red halo in 48 hours, persisting for several days and leaving a small pigmented nodule. The patient had an ulcer, 12 cm. in diameter, near the right tendo Achillis; biopsy showed the tissue changes of a leishmanial infection, but leishmania were not seen. The authors have studied the reaction in 23 leprosy patients and they think it is of possible value in the diagnosis of mixed infections.

H. Harold Scott.

PEREIRA, A. C. Lepra de forma clinica inaparente. [**Inapparent Leprosy.**] *Rev. Brasileira Leprologia.* S. Paulo. 1945, Dec., v. 13, No. 4, 269–72, 4 figs. on 2 pls. [11 refs.]

The case is recorded of a man, 20 years of age, giving no family history of leprosy, who, six months earlier had had on the dorsum of each foot raised, symmetrical, erythematous lesions, which were painful and slightly oedematous. Examination of his nasal mucus revealed the presence of Hansen's bacteria. Within a month the local lesions receded leaving no trace; nevertheless the nasal mucus, the inguinal gland juice and the lobules of the ears, which to all appearances were normal, showed the organisms. In spite of there being no clinical evidence of leprosy, the man was highly infective and all the more dangerous because of the absence of symptoms; he was, therefore, interned, but escaped soon afterwards.

H. Harold Scott.

DAVEY, T. F. **Some Observations on the Role of Allergy in Leprosy.** *Leprosy Review.* 1946, July, v. 17, No. 2, 42–62. [46 refs.]

This is a lengthy discussion, which is difficult to summarize, of what the author characterizes a hypothetical subject, because specific antibodies have not yet been proved in leprosy. Allergy is best seen in the tuberculoid form; and similarities with dermal conditions due to the tubercle bacillus are emphasized. The lepra bacillus has feeble pathogenic powers but it can act as an antigen. The early lepromin and the tuberculoid responses are distinct, but related, indications of the allergic state. In a sensitized tissue an effective concentration of antibody in response to the presence of bacilli is produced at an accelerated rate and some diffusion is probable. The young epithelioid cell is said to be the most effective bacteriological element in the tuberculoid response. The appearance of secondary spreading foci indicates a fall of antibody in the skin caused by a decline in sensitivity. The work of DHARMENDRA and LOWE indicate that the release of bacillary protein on breaking down of the lepra bacilli is a necessary preliminary to the allergic response, and a time-lag may be due to the interval required for this to take place. The antibody must be synthesized in the reticulo-endothelial system of the skin; this is a local reaction because one tuberculoid macule may resolve while another spreads at its edge. The author concludes that while allergy is not equivalent to immunity, a successful allergic response imparts a degree of local immunity as well as destroying the bacillus.

L. Rogers.

SCHUJMAN, S. Estudo comparativo entre a reação de Mantoux e a de Mitsuda nas diversas formas clínicas da lepra. [**Comparison of the Mantoux and Mitsuda Reactions in Leprosy.**] *Rev. Brasileira Leprologia.* S. Paulo. 1945, Dec., v. 13, No. 4, 231–6. English summary.

This paper deals with observations upon 210 adult patients suffering from leprosy in the city of Rosario.

The object of the investigation was to compare the results of the Mantoux and Mitsuda reactions in the nodular and the nervous-tuberculoid forms of leprosy respectively.

Initial grouping divided the patients into 122 cases of the nodular type (Mitsuda negative) and 88 of the tuberculoid type (Mitsuda positive).

After performing Mantoux tests on all the patients, the author arrived at the following conclusions :—

(1) The percentage of anergy to tuberculin (Mantoux negative 1/10) is higher in the nodular than in the tuberculoid type, namely 19 per cent. as compared with 3·5 per cent.

(2) The discrepancy arising from the result " Mantoux positive and Mitsuda negative " is only observed in nodular cases, while in tuberculoid cases, the two tests show agreement to the extent of producing 96 per cent. positive results.

(3) The discrepancy between the Mantoux and Mitsuda reactions in the nodular type of leprosy is explained by the existence of specific anergy to *Myco. leprae* on the part of patients suffering from this type of the disease.

H. J. O'D. Burke-Gaffney.

MONTEL, R. Aspects différents du bacille de Hansen dans le sang de leprome coloré par la méthode de Macchiavello. [**Hansen's Bacillus stained with Macchiavello's Method.**] *Bull. Soc. Path. Exot.* 1946, v. 39, Nos. 5/6, 167–70.

This method consists first of staining for two and a half minutes with a 0·25 per cent. solution of fuchsin, then differentiating by passing the slide very rapidly through a 0·5 per cent. solution of citric acid, and finally counterstaining for a few seconds with a 1 per cent. solution of methylene blue. Specimens of the leucocyte layer of citrated blood from a vein of a case of lepromatous leprosy with bundles of lepra bacilli in large monocytes, when stained in this way, showed large numbers of clear unstained bacilli seen against the blue background of the corpuscles. In addition a very few short bacilli, only one or two per leucocyte, were found stained vivid red ; as described in another paper by the same author [see the following abstract]. This finding raised some interesting questions regarding the significance of the very numerous unstained and the very few stained bacilli in such specimens. The author suggests that the unstained ones represent the usual form of Hansen's bacillus furnished with a waxy coat which prevents the action of this stain ; but the few red bacilli are young forms without a waxy coat, which take the stain. The former may not be able to infect, but the latter may be the only infective forms. Their rarity might account for the slight degree of contagiousness of leprosy.

L. Rogers.

MONTEL, R. Bacillémie lépreuse. Affinités tinctoriales du bacille de Hansen. [**Bacillaemia of Leprosy. Staining Characters of Hansen's Bacillus.**] *Bull. Acad. Méd.* 1946, v. 130, Nos. 9, 10 & 11, 165–8.

In leprosy the presence of the bacilli in blood should be determined by puncturing a vein through *unaffected* skin. He advises withdrawing 2 to 3 cc. in sodium citrate solution, and centrifuging. Smears should be made from

the layer of leucocytes, because the bacilli are nearly always found in bundles in large monocyte cells ; although a few may be free in the blood stream. By this method positive results nearly always are obtained in generalized lepromatous cases, especially during febrile reactions ; but never in the author's experience in tuberculoid and trophoneurotic cases. On staining the blood preparations by Macchiavello's method, faggots of bacilli may be seen unstained, in contrast to a few of a deep red colour in the cells [see the preceding abstract] ; the stained ones are very short. On staining with heated Giemsa for half an hour, the few short bacilli stain to a mallow or mauve colour. *L. Rogers.*

DE WILDEMAN, E. A propos de médicaments antilépreux d'origine végétale. VI. *Senecio* L. [**Vegetable Anti-Leprous Drugs.**] *Inst. Roy. Colonial Belge—Bull. des Séances.* 1946, v. 17, No. 1, 317–53.

In this long paper the author enumerates 103 species of the genus *Senecio* and the family of Compositae, together with notes of the medicinal uses to which they have been put. Alkaloids have been described in a number of them, the names of nine of which are given ; they appear to differ little among themselves and little or nothing seems to be known of their action in the numerous diseases of man and beast for which they have been recommended from time to time. Among the diseases of man referred to are cardiac, respiratory, liver, urinary affections and diseases of the male and female sexual organs. Among general diseases are hysteria, anthrax, syphilis, and a number of diseases affecting the skin, such as ulcers and boils, diabetes and leprosy. The only variety mentioned with regard to the last named is *Senecio Kleinia* of the Indies and Canary Islands. The author points out that little is known regarding the precise chemical constituents of these plants, some of which are highly toxic. Research is therefore required regarding their true toxic principles and their distribution in different parts of the plants. Until this has been done recommendations as yet made for their uses in medicine must be regarded as preliminary and provisional. The present paper will be useful to anyone undertaking a serious study of the medicinal properties of this genus. *L. Rogers.*

CALDEIRA, R. da Gloria. Contribuição a' terapeutica da reação leprótica. [**A Contribution to the Treatment of the Lepra Reaction.**] *Rev. Brasileira Leprologia.* S. Paulo. 1945, Dec., v. 13, No. 4, 277–9.

The author has used pituitrin successfully in the treatment of the lepra reaction.

SLOAN, N. R. **Tracheotomy in Leprosy.** *Hawaii Med. J.* 1946, Jan.–Feb., v. 5, No. 3, 125–8, 4 figs.

ROGERS, L. **Progress in the Control of Leprosy in the British Empire.** *Brit. Med. J.* 1946, June 1, 825–8.

This is a very full extract of a paper read before the Royal Society of Arts (*Roy. Soc. Arts.*, 1946, v. 94, 525) which deals with the general progress, during the last three decades, of prophylaxis against leprosy. Reference is first made to the introduction of the modified treatment by injection of suitable products of the old Indian remedy, chaulmoogra oils, with improved results in early cases only. Next, epidemiological work to enable a plan to be formulated whereby early cases may be detected by repeated examinations of close contacts of known cases of leprosy, with their treatment on a large scale at outpatient clinics. In addition it is essential to success in reducing the incidence

of leprosy to provide agricultural leprosaria for the admission and treatment of the advanced infective lepromatous cases, under a voluntary system as far as possible; the old rigid compulsory segregation must also be modified to allow of out-patient treatment of early cases, which are otherwise hidden. Successful trials of these methods, with the help of the British Empire Leprosy Relief Association, founded in 1924, are recorded. Only the fringe of the problem has yet been tackled, in view of the number of cases of leprosy in the British Empire, now estimated at two million; but the way has been opened up for extending gradually the use of the modern methods described. *L. Rogers.*

FIELDING, J. W. **Further Observations on Rat Leprosy.** *Med. J. Australia.* 1946, May 18, v. 1, No. 20, 681–6.

Further experiments on rat leprosy are recorded in this paper. Light natural infections with rat leprosy have been found in one *Rattus norvegicus* and in one *R. rattus*. Other rats are reported to have become infected from excreta containing acid-fast bacilli in the mud floors of their cages, after a few weeks of contact. Moreover, continued inunction with organisms in excreta results in "the production of intracellularity of organisms and in lesions," but there was no evidence of typical maximum intracellularity similar to that observed in leprotic lesions. It was also found that carbolized organisms were still viable and capable of undermining the resistance of an animal. Immunization was not obtained by injections of carbolized organisms. After removal from contact with infected soil, rats may become free from infection after 72 days. It is also stated that the infection of rats by rat leprosy is not enhanced by vitamin B_2 deficiency. [The claims made in this and the previous paper, see this *Bulletin*, 1945, v. 42, 903, are based on the assumption that the acid-fast bacilli found in the excreta of infected human beings and rats are the true causative organisms of leprotic infections.] *L. Rogers.*

HELMINTHIASIS.

BADIR, G. **Schistosomiasis of the Conjunctiva.** *Brit. J. Ophthalm.* 1946, Apr., v. 30, No. 4, 215–21, 3 figs. on 2 pls.

Schistosomiasis of the conjunctiva is reviewed by Badir and he describes a case which is unique in that it is the first one reported where the worms were observed *in situ* under the conjunctiva. The patient was a boy aged 12 years; on examination, a tumour of the palpebral conjunctiva of the left upper eyelid was found near the inner canthus. The tumour had been present for more than a month. The swelling was excised and the specimen on examination showed a large number of terminal-spined bilharzia ova [presumably *S. haematobium*]. Many of the ova were degenerated. A male and an included female bilharzia worm were seen lying in a dilated orbital vein.

E. O'G. Kirwan.

KAMEL, A. **Schistosomiasis (Bilharziasis) of the Conjunctiva.** *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 118–23.

In spite of the fact that Egypt is highly infested with bilharzia and that bilharzial granulomata have been found in nearly every part of the body, yet bilharzial lesions of the eye are very rare. The ova reach the conjunctiva probably during the migration of young worms; some, to reach their goal, probably wander the wrong way into the vessels of the head to find themselves

in the vicinity of the mucous membrane of the conjunctiva, where they deposit their ova. The ova deposited in the submucosa are seen to be surrounded by an area of digestive rarefication produced by digestive enzymes secreted by the lateral glands of the embryo. To this zone of rarefaction leucocytes migrate, and the eosinophil cells are the most prominent. This is what is called "schistosomiasis pseudo-abscess" and differs from the ordinary pyogenic abscess in that it does not suppurate.

The lesions may be seen in the conjunctiva as: (1) small yellowish-pink nodules on the bulbar conjunctiva, single or multiple, surrounded by leashes of small vessels and bearing great resemblance to streptothrix nodules of the conjunctiva; (2) polypoid masses arising from the fornices; (3) chalazion-like granulomata on the tarsal conjunctiva. In all forms, however, one can easily recognize yellowish dots in the centre, not unlike the post-trachomatous degenerations of trachoma, and if one ruptures and spreads the contents of such nodules on a slide, ova can be found by direct examination under the microscope. The lesions are not painful and the patient's attention is usually attracted by the disfigurement. Unlike tuberculosis, the preauricular and submaxillary glands are not enlarged in schistosomiasis of the conjunctiva.

One case was seen in which the lesion was bilateral, and cases have been reported from Upper as well as from Lower Egypt. *E. O'G. Kirwan.*

ABDALLAH, A. **Bacteriological Flora in Urinary Schistosomiasis. A Report on 150 Cases.** *J. Roy. Egyptian Med. Ass.* 1946, Jan.–Feb., v. 29, Nos. 1/2, 33–7.

FLOCH, H. & DE LAJUDIE, P. Sur les bilharzioses en Guyane française. [**Schistosomiasis in French Guiana.**] *Institut Pasteur de la Guyane et du Territoire de l'Inini. Publication No. 119.* 1945, Dec., 5 pp. [14 refs.]

The authors make the point that most, if not all, of the few cases of *S. mansoni* infection which have been reported from French Guiana have been acquired outside that country. This infection is endemic in the Antilles, Venezuela, British and Dutch Guiana and elsewhere. *S. haematobium* infection, on the other hand, is not endemic anywhere in the Americas, and only imported cases have been seen. Schistosomiasis is not, therefore, an important disease in French Guiana, and the authors have failed to find the common American mollusc host, *Australorbis glabratus.* *Charles Wilcocks.*

WELLER, T. H. & DAMMIN, G. J. **The Incidence and Distribution of *Schistosoma mansoni* and other Helminths in Puerto Rico.** *Puerto Rico J. Pub. Health & Trop. Med.* 1945, Dec., v. 21, No. 2, 125–47, 6 figs. [Refs. in footnotes.] [Spanish version 148–65.]

"A single stool specimen from each of 19,139 Puerto Rican Selective Service registrants was examined for helminth eggs and larvae by a modification of the Telemann acid-ether centrifugation technique. While the group studied was not representative of the total adult male population, these findings comprise the first available data on a relatively uniform group of individuals drawn from all sections of the Island.

"Helminth eggs and larvae were found as follows: *Schistosoma mansoni* in 1,909, or 9·97 per cent.; Hookworm in 10,822, or 56·5 per cent.; *Ascaris lumbricoides* in 1,288, or 6·7 per cent.; *Trichuris trichiura* in 14,614, or 76·3 per cent.; *Strongyloides stercoralis* in 1,984, or 10·4 per cent.; *Hymenolepis nana* in 29, or 0·15 per cent.; and *Taenia* sp. in 5, or 0·03 per cent. The helminth index was 1·6.

" The data were analyzed to obtain information on the relationship of the occurrence of the five common parasites to the age and geographical and occupational distribution of the selectees."

BARROS, J. de R. Determinações nervosas da parasitose sanguínea de Manson e Pirajá da Silva. Forma epiléptica. [**Nervous Symptoms in Infestation by** *Schistosoma mansoni*.] *Publicações Médicas.* São Paulo. 1946, Feb., v. 17, No. 7 (159), 43, 45–8.

The author states that *Schistosoma mansoni* may give rise to symptoms of involvement of the nervous system varying from general irritability to attacks of vertigo and actual epileptic seizures. He has notes of " some thirty such cases " and gives details of one. The patient was a man of 29 years, seen by the author in 1937. He had had three attacks of malaria since the age of 12, but was generally healthy and strong ; there was no evidence of any venereal disease, nevertheless his attacks of abdominal and sacro-lumbar pain, occasional passage of stools with mucus and streaks of blood, vertigo, and nocturnal epileptic seizures with loss of consciousness were ascribed to syphilis and he was given antisyphilitic treatment, but without any benefit. Examination of the faeces showed " cysts of amoeba coli and ova of ankylostomes and of *Sch. mansoni*." After a course of thymol and of fouadin, he had no more vertigo or epileptiform attacks and he has remained well. He was last examined in 1945, 8½ years after coming under the author's observation.

H. Harold Scott.

HERNÁNDEZ MORALES, F. & OLIVER GONZÁLEZ, J. **Ova of** *Schistosoma mansoni* **in Purged and Unpurged Fecal Specimens.** *Puerto Rico J. Pub. Health & Trop. Med.* 1945, Dec., v. 21, No. 2, 209–10. [Spanish version 211–12.]

The authors examined the purged and unpurged faecal specimens of 120 persons, found to harbour *S. mansoni* ova in their stools after a dose of magnesium sulphate. The first faecal samples, when no purge had been given, revealed that ova could only be found in 58·3 per cent. of the patients. It is concluded that the ova of this parasite may be found more readily after the administration of a purgative.

TORRES, C. M. & PINTO, C. Lesões produzidas pelo " Schistosoma mansoni " no tatú (*Euphractus sexcinctus*), mecanismo de eliminaçao dos ovos e sensibilidade da espécie animal nas infestações experimentais. [**Lesions produced by** *S. mansoni* **in the Armadillo : Mechanism of Elimination of Eggs : Susceptibility to the Armadillo to Experimental Infection.**] *Mem. Inst. Oswaldo Cruz.* 1945, Oct., v. 43, No. 2, 301–48, 38 figs. (3 coloured). [23 refs.] [Summary in English by the authors.]

" 1. The post-mortem examination of a male armadillo (*Euphractes sexcinctus*) experimentally infected by *Schistosoma mansoni* has shown 86 adult female worms and 23 male worms inside the liver, and 4 (2 male and 2 female worms) inside the pancreas.

" Ova of *S. mansoni* were found in the faeces since the 53rd day after infection, persisting till the death of the animal 41 days later.

" Schistosomal pseudotubercles are very numerous in the small and large intestine, in the liver and haemolymph nodes. They could not be detected in the lungs, pancreas, heart-muscle, lymph-nodes, kidneys, bladder and testes.

" 2. Neither cirrhosis of the liver, nor polypoid endarteritis and endophlebitis could be demonstrated.

"3. In the small intestine, ova and schistosomal pseudotubercles remained strictly localized in the submucosa, and none of them was found above the *muscularis mucosae* (i.e. in the mucous coat itself). The small intestine, therefore, was not related in this particular animal to the elimination of the *S. mansoni* eggs, at least at the time in which the post-mortem examination was performed.

"4. In the large intestine, on the contrary, ova and schistosomal pseudotubercles were very numerous in the mucous coat itself, and this portion of the gastro-intestinal tract was actually concerned with a very active and widespread liberation of *S. mansoni* eggs.

"5. The chief factors affecting the liberation of *S. mansoni* ova as observed in this armadillo are, in our opinion: (1)—the structure of the large intestine provided with a less regular *muscularis mucosae* as compared with that of the small intestine, and larger blood vessels which enter the mucous coat; (2)—the extrusion of eggs in the capillaries according to the manner evidenced by Koppisch (1937) and their transient fixation in the mucous coat; (3)—the formation of a cellular infiltrate about the extruded ova; (4)—the histolysis of this cellular infiltrate as well as of the surrounding tissue; (5)—disintegration of the walls of the adjoining Lieberkühn's glands as the histolysis increases, and consecutive transfer of the eggs to the Lieberkühn's crypt; (6)—their further elimination in conjunction with the intestinal juice secreted by the glands.

"6. In the small intestine there is an acute catarrhal enteritis probably incited by bacteria or by toxic substances, besides a chronic enteritis associated with schistosomal pseudotubercles and eggs.

"7. In the large intestine there is a chronic catarrhal colitis associated with schistosomal pseudotubercles and ova, as well as early stages in the development of mucous polyps."

[For another note on the same subject, by the same authors, see this *Bulletin*, 1946, v. 43, 658.]

Mason, P. K., Daniels, W. B., Paddock, F. K. & Gordon, H. H. **Schistosomiasis Japonica. Diagnosis and Treatment in American Soldiers.** *New England J. of Med.* 1946, Aug. 8, v. 235, No. 6, 179–82. [10 refs.]

"Four hundred and eighty-one cases of schistosomiasis japonica acquired on Leyte, an island in the Philippines, were studied at an Army general hospital in the United States.

"Recurrent epigastric cramps and tenderness were the most frequent complaints on admission, but in almost all cases these had disappeared by the time of discharge.

"Repeated stool examinations by more than one method were necessary to demonstrate the eggs of *Schistosoma japonicum*. A persistent eosinophilia was suggestive but not diagnostic of continued activity of the disease.

"On the basis of comparable series, treatment with tartar emetic was found to be much more effective than that with fuadin. Significant toxic reactions with tartar emetic were rare.

"It is urged that stool examinations be done repeatedly and by multiple methods on men who have been exposed to this disease so that treatment may be instituted if ova are found."

Naval Medical Research Institute and U.S. Naval Hospital. Bethesda, Maryland. 1946, Apr. 29, Report No. 1. **Biological Studies of Antimony Compounds containing Radioactive Isotopes: III. The Blood-Tissue Exchange and Excretion of Antimony in Humans given a Single Dose of Tartar Emetic** [Smith, R. E., Stormont, R. T., Bianco, A. A. & Evans, R. L.]. 10 pp., with Appendix and 3 figs.

HALAWANI, A. & NOR-EL-DIN, G. **Encephalopathy following Treatment of Schistosomiasis with Tartar Emetic.** *J. Roy. Egyptian Med. Ass.* 1946, Jan. & Feb., v. 29, Nos. 1/2, 76–9.

TUCKER, H. A. **Intestinal Cestode Infections in Natives of Panama.** *Puerto Rico J. Pub. Health & Trop. Med.* 1946, June, v. 21, No. 4, 364–5. [Spanish version 366–8.]

"1. In a review of 558,566 admissions to Gorgas Hospital, Ancon, Canal Zone, from May 1904 to November 1944, 126 instances of intestinal cestode infections were found. Only 15 of these were unquestionably autochthonous in origin.

"2. The paucity of suitable intermediate hosts, together with inspection and thorough cooking of meats, were considered the most important factors in explaining this low incidence.

"3. All parasites found were generally accepted as being cosmopolitan in distribution; only 3 species could be definitely considered of local origin—*Taenia saginata, Taenia solium,* and *Hymenolepis nana.*"

HERNÁNDEZ MORALES, F. **Poisoning by Oleoresin of Aspidium. Report of a Case with Post Mortem Findings.** *Puerto Rico J. Pub. Health & Trop. Med.* 1945, Dec., v. 21, No. 2, 213–18. [Spanish version 219–25.]

LATIF, N. & EL KORDY, M. I. **On the Vitamin Content of Hydatid Fluid.** *J. Roy. Egyptian Med. Ass.* 1946, Jan.–Feb., v. 29, Nos. 1/2, 71–5.

DA SILVA, P. B. Estrongiloidíase. Sintomatologia e tratamento. [**Strongyloidosis. Symptoms and Treatment.**] *Publicações Médicas.* São Paulo. 1946, Feb., v. 17, No. 7 (159), 49, 51–2.

In 1937 the author had occasion to pass a duodenal tube on a man, 23 years of age, with signs of duodenal ulcer and he found in the bile and duodenal material abstracted a large number of ova and larvae of *Strongyloides stercoralis.* He was accustomed to treat gastric and duodenal ulcers by daily lavage with 0·1 per cent. gentian violet, and gave that treatment to this patient. At the end of a week no more worms were seen and the patient felt well.

Some days later, a girl of 11 years was seen, suffering from severe anaemia and obstinate diarrhoea; the faeces contained larvae of *Strongyloides* in large numbers. She objected to the passage of a duodenal tube, so the gentian violet was given twice daily in capsules, each containing 0·025 gm., for 10 consecutive days. The diarrhoea ceased and the anaemia had improved and examination of the stools failed to reveal any worms. A brother of the last, aged 8 years, similarly affected, was cured in the same length of time.

In his conclusions the author states that infestation by *Strongyloides stercoralis* is fairly common in Rio State; in the course of about 2,000 duodenal intubations he has met with an average of 30 per cent. infested; that the best results in treatment are attained by the introduction of 20 cc. of a 0·1 per cent. solution of gentian violet by means of a duodenal tube, or by capsules of 0·025 gm. of the drug twice a day. If the patient complains of giddiness or malaise after taking the drug, it should be given after the chief meals of the day.

H. Harold Scott.

LANDSBOROUGH, D. **Ascariasis causing Acute Intestinal Obstruction.** [Memoranda.] *Brit. Med. J.* 1946, Sept. 28, 461.

PEEL, E. & CHARDOME, M. Note préliminaire. Sur des filaridés de Chimpanzés, *Pan paniscus* et *Pan satyrus* au Congo Belge. [**A Preliminary Note on Filarial Worms in Chimpanzees in the Belgian Congo.**] *Rec. Travaux Sci. Méd. Congo Belge.* 1946, May, No. 5, 244–7, 4 figs.

The authors report the finding of the adult female *Agamofilaria streptocerca* in *Pan paniscus*; full details will be published later.

PACHECO-LUNA, R. **Notes on Oncocerciasis in Guatemala.** *Brit. J. Ophthalm.* 1946, Apr., v. 30, No. 4, 234–7. [19 refs.]

[The author spells Onchocerciasis "Oncocerciasis" throughout.] Onchocerciasis in Guatemala is described by Pacheco-Luna. It was discovered there in 1915 by ROBLES and is endemic principally in regions devoted to the production of coffee and situated at altitudes of 300 to 1,200 metres, on the steep slopes of the Sierra Madre. It is estimated that 20,000 persons are infected. Ocular manifestations are observed in 30 per cent. of these, and 2 per cent. are blind. The signs of onchocerciasis are classified under three heads:—the tumours; the characteristic ocular signs which sometimes end in blindness; and some rare cutaneous manifestations. The ocular changes are due to a chronic, slow and invidious process of sclerosis, which takes years to develop. It is caused by the presence of the microfilariae, alive or dead, in the tissues of the eye, and by the secretions and excretions of both microfilariae and filariae. These act both locally and at a distance, possibly by means of toxic products of protein disintegration. The early symptoms are severe photophobia, blepharospasm and a sensation of a foreign body in the eye. At first only a slight ciliary injection is visible, but with the corneal microscope a superficial punctate keratitis can be seen. This affects the ends of the horizontal diameter, leaving the centre of the cornea free. Later the infiltrations invade the deeper tissues. In some cases, whilst the keratitis is in progress, but in others independently, a plastic iritis develops which seriously affects vision. At first the pupil is contracted. Synechiae and pseudo-membranes develop which obstruct the pupil and force it downwards, giving it a pear-shape. The process extends to the uveal tract, and degenerated pigmentary lesions of the choroid and retina have been observed. Examination of them is difficult owing to the clouding of the vitreous. In advanced cases, the ocular tension is low and the process ends by *phthisis bulbi*. Unfortunately, up to the present, no therapeutic agent has been found which acts with good results upon the parasites.

E. O'G. Kirwan.

DEFICIENCY DISEASES.

CRUICKSHANK, E. K. **Painful Feet in Prisoners-of-War in the Far East. Review of 500 Cases.** *Lancet.* 1946, Sept. 14, 369–72, 2 figs.

The author saw 500 cases of this syndrome among British and Australian prisoners in Changi Camp, Singapore. It appeared after six months of captivity, during which the prisoners had received a diet supplying adequate calories but deficient in first-class protein and in the B group of vitamins. Fresh cases ceased to appear in any numbers, and the sufferers gradually recovered, after a few months when there was an all-round improvement in the diet, although the quantity of riboflavin supplied remained low. The greatest incidence of painful feet occurred at a time when fresh cases of recognized thiamin deficiency were relatively few.

In early cases, the general condition was good unless there had been some recent debilitating disease, such as dysentery or malaria, which were considered common precursors of the syndrome. The general condition tended to deteriorate as the syndrome became chronic, partly from lack of sleep and the continual pain. The syndrome usually started with a dull ache below the heads of the metatarsal bones and in the balls of the toes. This pain became more pronounced and almost continuous ; rather more than half of the sufferers then had sharp stabbing pains superimposed on the ache. Both pains were worse when the patients went to bed. A few patients had similar pains in the arms.

Inspection of the legs and feet revealed nothing that could not be demonstrated equally in the other prisoners. Hypersensitivity to light touch was present in just under a quarter of the patients, but there was no deep tenderness. Rather less than a quarter of the patients showed exaggerated tendon reflexes in arms and legs. The blood pressure was recorded in 189 cases, and 51 of these had pressures above 130/90 mm. Hg. The symptoms of all those with high blood pressure were severe and of long standing. Except in one man, who was believed to have essential hypertension, these pressures all returned to normal as the syndrome regressed.

Intravenous thiamin given to six men in doses of 2 mgm. daily for a week, gave no improvement. No good effect was observed after giving nicotinamide by mouth ; intramuscular injections produced improvement, but the best and most rapid results were given by intravenous therapy. Nevertheless, some patients showed no improvement after a course of intravenous nicotinamide. Marmite, rice polishings and boiled green gram by mouth appeared to have some favourable action, but in the absence of controls this is not certain.

H. E. Harding.

GOPALAN, C. **Some not well-known Manifestations of Riboflavin Deficiency.** *Indian Med. Gaz.* 1946, June–July, v. 81, Nos. 6/7, 227–30.

"In this paper some manifestations of riboflavin deficiency which have received inadequate attention, *viz*, leucorrhoea, fissure of the anus, angular blepharitis and prepucial ulceration, are described."

WOOLLEY, D. W. **The Occurrence of a "Pellagragenic" Agent in Corn.** [Correspondence.] *J. Biol. Chem.* 1946, June, v. 163, No. 3, 773–4.

Mice were given a basal diet of sucrose, casein, cystine, salts, fortified corn oil, and adequate water soluble vitamins except nicotinic acid. The addition of a chloroform-NaOH extract of maize to this diet resulted in considerable lessening of the growth rate : in addition, many of the animals developed mild diarrhoea and considerable reddening of the skin and tongue. This effect of the maize-extract was prevented if nicotinamide was also added to the basal diet.

The substance responsible for these effects appeared to be a weak base, soluble in water. It could be extracted from the chloroform solution by dilute H_2SO_4, and remained in aqueous solution after precipitation of sulphate by barium hydroxide. Its nature has not been further determined.

H. E. Harding.

SPRUE.

SMART, G. A. & DALEY, R. **Fat-Digestion in Sprue studied by Intestinal Intubation.** *Lancet.* 1946, Aug. 3, 159–60, 1 fig.

Preliminary investigations are here presented on ten subjects invalided to England with tropical sprue originally contracted in India, Burma or Ceylon.

Eight patients who had never been in the tropics acted as controls. Their preparation is described in detail. After preliminary fast, a radio-opaque, double-lumen, Miller-Abbott tube, guided by radioscopy, was passed into the small intestine and swallowed up to the 72 in. mark, when the tip was 40 in. beyond the pylorus. The balloon was then deflated. After an interval of 30 minutes, suction of 100 mm. Hg was applied to the lumen of the tube, and the material was collected in a bottle surrounded by ice. After the fasting juice had been thus obtained for about 20 minutes, the patient swallowed 25 ml. of olive oil. Suction was continued and, whenever any alteration was noted in the character of the aspirated material, the bottle was changed. Fat began to appear in 30–90 minutes after it had been swallowed, and specimens were collected until they no longer contained it.

The material aspirated from sprue patients differed strikingly from that of the controls; in the former it consisted of whitish threads and clumps in a relatively clear bile-stained fluid; in the latter it was a uniformly suspended bile-stained emulsion.

On microscopic examination these threads and clumps contained fat droplets enmeshed in mucin. Similar, but smaller, droplets were seen in normal patients, but they were evenly dispersed. After storage for 24 hours at 0°C. the droplets from the patients with sprue and from the controls stained blue with Nile blue sulphate, indicating the presence of fatty acids. Only a small proportion consisted of unsplit fat and stained pink.

The extent of the abnormality varied in different patients. In one, this examination was carried out before and after successful treatment by diet and parenteral crude liver extract, when the character of the aspirated material changed. It would therefore appear that in the sprue syndrome fat is emulsified and, as would be expected, hydrolyzed. With the object of ascertaining the possibility of dispersing these fatty aggregations, specimens from three patients were incubated at 37°C. for four hours with fasting succus entericus.

Good dispersal was obtained with normal patients, but none was obtained in one mild and one severe case of sprue. The addition of ox-bile aided the dispersal to some extent in two other cases. The evidence obtained appears to suggest that the main factor is the presence of mucin, which is more resistant than proteins to the digestive action of the proteolytic enzymes. It is probable that mucus assists in the emulsification of fat, and it is possible that, by adsorbing lipase, mucus also assists in its hydrolysis.

In sprue it appears that mucin is not digested and, thus, interference with the absorption of fatty acids results. *P. Manson-Bahr.*

FRAZER, A. C. **Normal and Defective Fat Absorption in Man.** *Schweiz. med. Woch.* 1946, v. 76, Nos. 37/38, 903–8.

BLACK, D. A. K., FOURMAN, P. & TRINDER, P. **The Faeces in Sprue.** *Brit. Med. J.* 1946, Sept. 21, 418–20.

This is a record of part of a G.H.Q. (I) investigation on tropical sprue. Examinations of 500 four-day specimens of sprue stools were made on patients with proven impaired absorption of fat and clinical manifestations of sprue. The patients were on a diet containing either 66 gm. or 96 gm. of fat daily; the stools were collected and weighed in bulk before determination of total fat, fatty acid and soap contents; the water content and non-fat dry residue were calculated from the weighings, and the *p*H was roughly determined by indicator strips. The majority of the stools conformed to the classical sprue type, but

two important deviations from this were encountered, *viz.*—(1) Many stools with a high fat content were well pigmented and not unduly bulky—an observation previously noted [Black and Fourman, this *Bulletin*, 1946, v. 43, 474] : further, on exposure to light and air pale stools darken in colour ; and (2) many patients with sprue suffer from attacks of watery diarrhoea (clinical dysentery). The lack of pigmentation in the sprue stool is usually held to be due to the reduction of stercobilin to stercobilinogen, but simple dilution of the pigment by fat may be contributory to it. The dysenteric diarrhoea may be due to an intercurrent bacillary dysenteric infection, in which case it responds to sulphaguanidine treatment, but, possibly more often, sulphonamide-susceptible organisms, normally commensal, become pathogenic in an abnormal gut. Such diarrhoea was observed to yield promptly to parenterally-administered liver-extract.

The ratio of split to unsplit fat in sprue usually exceeds the normal 3 : 1 or 4 : 1, and this has been attributed to defective absorption of split as opposed to unsplit fat. Bloor (*Biochemistry of the Fatty Acids*, New York, 1943) found that normal stools can partially break down neutral fat added to them ; the authors find that this lipolytic property is possessed in even greater degree by sprue stools, probably because of the emulsifying action of their high soap content. The lipolytic action was not arrested by penicillin or sulphathiazole, so is not bacterial in origin ; it was lessened by the addition of 5 per cent. copper sulphate. The relative absorptions of split and of unsplit fats in sprue, therefore, cannot be deduced from an analysis of the stools.

The ratios of total split fat and of split fat in the form of soaps in 46 dried stools from ten patients are set out in tabular form. In all but six of these the ratio of split fat present as soaps was found to be over 60 per cent., irrespective of the total split fat content. Those stools with the greatest content of split fat, and containing the greatest amount of soluble soap, were the most alkaline —which suggests that the irritant nature of very fatty stools is due to a high content of soluble soaps and not to acidity.

The non-fat dry residue (N.F.D.R.) is composed largely of bacteria and of unabsorbed food residue. On a controlled diet this residue should remain constant ; but the authors found that after treatment of many, but not all, of their sprue cases with liver and yeast-extract, the N.F.D.R. diminished. The cause of this was not determined, but such a diminution in this residue may mask a decrease in the gross fat excretion, the percentage figure for fat in the dried stool as a result of it being unaltered.

With gross variations in steatorrhoea, such as occur in sprue under treatment, a fair measure of the water content of the stool cannot be determined by the ratio of dry to wet stool weight, the fatty part of the stool not being wetted. A " percentage water content " has been calculated from wet weight of stool minus fat and dry weight of stool minus fat, and average figures for 66 stools from 11 patients are given ; stools of higher fat content were found to have a higher percentage of water.

In mild or atypical sprue the percentage of fat in the dried stool may be normal, especially if the patient is restricting his fat intake. To demonstrate steatorrhoea such patients must be put on a diet containing 90–100 gm. of fat daily and the stools collected for at least 24 hours, and preferably for four days. The fat excreted on such a diet should not exceed 10 gm. daily in a normal person. Where facilities for collecting and weighing stools are lacking, a percentage of estimation of fat in a mixed 24-hour specimen is of more value than an observation on one stool, and a high residue diet must be avoided so as not to mask minor degrees of steatorrhoea. For accurate observations on fat excretion, twelve-day periods of observation are a minimum requisite.

A. R. D. Adams.

FRANDSEN, S. & KRINGELBACH, J. **Investigations in the Behaviour of the Serum Iron in Hyperchromic Sprue Anemia.** *Acta Med. Scandinavica.* 1946, v. 125, No. 1, 29–39, 2 figs. [10 refs.]

By the term "sprue" in this article the form more generally known as idiopathic steatorrhoea is probably indicated.

Two cases of untreated hyperchromic anaemia in sprue which were investigated immediately after injections of liver showed a reduction of the serum-iron value from high normal values to low subnormal values, as well as a tendency to persistent low values during continued treatment. The curve thus obtained corresponds to that usually observed in the serum-iron of pernicious anaemia during the course of similar treatment. In the first, during a reticulocyte rise the peak was reached just after a decrease of the serum-iron content had been noted. In the second case, decrease in serum-iron was more protracted.

In these instances, where similarity of the behaviour of serum-iron during the liver treatment of hyperchromic sprue anaemia and that of pernicious anaemia is observed, a certain parallelism is to be expected. It appears that no prediction as to the eventual response of the anaemia to specific treatment can be made from the behaviour of the serum-iron. In these two cases as a result of treatment, the serum-iron fell from normal (high) values—152–179γ per cent. and 173–152γ per cent. to subnormal values—68γ per cent and 37γ per cent.—respectively, analogous to the conditions observed in pernicious anaemia.

P. Manson-Bahr.

WINGFIELD, A. L. **Some Observations on the Aetiology and Treatment of Sprue,** *Proc. Roy. Soc. Med.* 1946, July, v. 39, No. 9, 519–22 (Sect. of Med. 15–18). 2 figs. [14 refs.]

The author believes that the very rapid response to treatment points to a disorder of function rather than of structure as the basis of sprue. Since 1939 he has made it his practice to permit all patients with sprue to partake of full ward diet and to get up when they wish. They are treated with crude liver injections (Plexan), 4 cc. daily for one week, then on alternate days for one week and twice weekly thereafter; and to this is added nicotinic acid, 50 mgm. three times daily.

"A single dose of nicotinic acid, 150 mgs., is given to test vasomotor and digestive reactions. So far, no patient tested has complained of flushing before the beginning of treatments, while all patients have given a specific vasomotor response after treatment, for two to three weeks. This observation supports the contention that crude liver quickly restores intestinal function." Nine patients were treated on these lines with satisfactory response; the average gain in weight has been 27 pounds, varying from 13–52 pounds. Stools return to normal in about seven days. Untoward phenomena have been pain at site of injection, and transient oedema of the legs and feet has occurred in the second week, possibly because of sodium retention.

One extreme case is cited; the patient on this mode of treatment, doubled her weight.

His experiences have led the author to believe that diet is of little or no consequence in the treatment of sprue. Crude liver extract is important, possibly because it is a potent source of vitamin B_2 complex, but it also contains an unidentified fraction which may prove to be the specific factor.

P. Manson-Bahr.

HAEMATOLOGY.

CASE, R. A. M. **Siderocytes in Mammalian Blood.** *Proc. Roy. Soc.* Ser. B. 1946, Aug. 7, v. 133, No. 872, 235–48, 7 figs. [21 refs.]

" 1. The siderocyte appears in large numbers in stored blood and in blood that has been treated with certain chemical agents. Phenylhydrazine is such an agent.

" 2. Apparently all erythrocytes in these experiments go through a stage as a siderocyte, but the granules are extruded, leaving a cell which is at present morphologically indistinguishable from a pre-siderocyte erythrocyte.

" 3. As siderocytes have now been demonstrated in mouse, rat and man (Grüneberg 1941 *a*, *b*) ; in guinea-pig, horse and rabbit (Granick 1943) and in cat and dog (Case 1943), the cell is probably to be found in all mammals.

" 4. After an erythrocyte has become a siderocyte it becomes susceptible of phagocytosis, and is removed from the circulation in the intact animal.

" 5. The stainable non-haematin iron is derived from a special fraction of the haemoglobin, and the other side of the breakdown is bile pigment. Therefore, the iron should be regarded as catabolic, and closely associated with, if not identical with, the E.S.I. described by other workers.

" 6. The siderocyte is probably the source of the urinary siderotic granules described by Rous (1918).

" 7. The siderocyte probably has importance in clinical medicine."

ROMIJN, C. De klinische haemoglobinebepaling, getoetst aan enkele laboratoriummethoden. Eerste mededeeling: De klinische haemoglobinebepaling. [**The Clinical Estimation of Haemoglobin.**] *Tijdschr. v. Diergeneesk.* 1946, Sept. 1, v. 71, No. 17, 688–97, 1 fig. [18 refs.] [Summary in English by the author.]

" A critical review of the methods of haemoglobin estimation by means of the s.c. acid haematin method has been given. The conversion of oxyhaemoglobin into acid haematin is a reaction with a velocity which varies from individual to individual. Haemoglobin-determinations with the s.c. Zeiss-Ikon haemometer and with a laboratory method were carried out with blood of several horses and cows. The differences between the results with the clinical haemometer and with the precision method (calculated from the oxygen capacity or spectrophotometrically) cannot be eliminated by correction of the standard of the apparatus.

" With the Sicca-haemometer from Dr. PHILIPSEN (Vedbaek, Denmark) much better results were obtained. When this instrument is standardised on a precision method (spectrophotometric method), the results obtained with the Danish haemometer agree very closely with the haemoglobin readings made with a good laboratory method. The advantages of this instrument over the acid haematin method are twofold : first, undiluted blood is used and further there is no " time factor " in reading the haemoglobin content of the blood. In the present time the standard of the Sicca-haemometer is adjusted on about 16 grm.% Hb, corresponding with 100% of the scale."

RAPPAPORT, F. & EICHHORN, F. **A Simplified Hematocrit.** *Harefuah.* Jerusalem. 1946, Sept. 1, v. 31, No. 5. [In Hebrew 80, 1 fig. English summary 80–81.]

STEVENSON, I. P. **Sickle Cell Anaemia.** *Arizona Med.* 1946, May, v. 3, No. 3, 161–3.

ALTMANN, A. **Cooley's (Mediterranean) Anaemia in a South African-Born European Adolescent.** *South African Med. J.* 1946, Aug. 24, v. 20, No. 16, 476–8, 1 fig. [21 refs.]

" A case of Cooley's anaemia (Mediterranean) is described in a South African youth with no known relation to Mediterranean races (except for a French great-grandmother). The condition is characterised by a mild anaemia with hypochromia, aniso- and poikilocytosis of the R.B.C.s, increased blood destruction and increased blood regeneration. The resistance of the R.B.C.s to haemolysis in hypotonic saline is markedly increased."

DERMATOLOGY AND FUNGUS DISEASES.

FERGUSSON, A. G. **Skin Diseases in the Tropics.** *Practitioner.* 1946, Aug., v. 157, No. 928, 114–22.

All the common vascular disorders of the skin are frequently encountered in the tropics. Urticaria is one of the commonest, and the aetiological factors which have to be considered—besides those usually recognized in Britain—are heat and parasitic infestations such as taeniasis, ascariasis, ancylostomiasis and amoebiasis ; appropriate measures must be taken to combat these and other causes of the malady.

The secondary pyococcal infections which so often complicate the exudative dermatoses, are facilitated by excessive sweating, which causes the sweat to become relatively alkaline, and to become a suitable nidus for the proliferation of pathogenic bacteria.

For the protection of skin which is sensitized to sunlight, Fergusson advocates the use of a cream containing quinine, ichthyol, or disodium naphthol sulphonate, for all of these drugs retard or prevent the access of ultra-violet rays to the integument. Resorcin is the best protective against luminous rays ; URBACH and KONRAD (*Strahlentherapie*, 1929, v. 32, 193) have suggested the use of zinc paste loaded with 3 per cent. resorcin for this purpose.

Psoriasis can be very troublesome in hot countries where " any benefit derived from sunlight . . . is usually more than offset by the deleterious results of excessive sweating and secondary pyogenic infection. Further, the disease appears to be quite common among the scantily clothed natives of tropical regions." Owing to the macerating effects of greasy ointments and creams, paints and lotions are more commonly used than at home. In the acute stage of psoriasis an emulsion containing 2 per cent. of sulphur is useful, whilst the malady when indolent can be treated by applications of 20 per cent. crude coal tar in acetone, or by cignolin (dithranol) ½–2 per cent. in the same vehicle. Tar and ultra-violet light baths, given twice or thrice weekly, are helpful in resistant cases.

The diseases of the seborrhoeic group are often troublesome and are aggravated by heat and sweating. Seborrhoeic subjects are " bad risks " in hot countries, and should spend as much time as possible in dry and cool areas. Whenever possible patients with seborrhoeic dermatitis should be evacuated to cooler zones, *e.g.*, hill stations. Fergusson has found that injections of penicillin (15,000 units every three hours until 120,000 units have been given) are satisfactory in combating the secondary infections which often complicate these maladies.

Mild cases of ichthyosis improve in the tropics ; severe cases are usually aggravated and may readily develop heat exhaustion.

Of mycotic infections, the author states that small-spored ringworm of the scalp is often seen in natives, particularly Ghurkas. An aqueous solution of 10 per cent. sodium hexa-metaphosphate applied twice daily to the feet controls sweating and is a useful prophylactic against tinea pedis.

The " desert sore " and the " jungle sore " of the recent war were manifestations of ecthyma ; secondary infections of the lesions with diphtheria bacilli may occur if faucial or nasal diphtheria is prevalent. The most satisfactory treatment of ecthyma is the frequent application of a penicillin spray containing 500 units of the antibiotic in each cc. of saline.

The paper concludes with a reference to prickly heat ; this malady is best treated by avoiding unnecessary exertion, stimulating foods and alcohol, by wearing light clothing, taking a tepid bath twice daily and applying LEWIS's dusting powder [this *Bulletin*, 1942, v. 39, 648] freely ; this powder contains equal parts of zinc, starch, boric acid and sulphur. *R. M. B. MacKenna.*

SULZBERGER, Marion B., ADDENBROOKE, E. F., JOYCE, S. J., GREENBERG, S. & MACK, A. G. **Tropical Acne.** *U.S. Nav. Med. Bull.* 1946, Aug., v. 46, No. 8, 1178–84, 2 figs. Also in *Bull. U.S. Army Med. Dept.* 1946, Aug., v. 6, No. 2, 149–54, 4 figs.

The authors noted—and have recorded their findings in two journals—that in military hospitals on the island of Guam, 10 per cent. of all the cases admitted were suffering from skin diseases ; further, 60–70 per cent. of all men attending sick parades did so because of cutaneous maladies. Approximately 20 per cent. of these dermatological cases were diagnosed as " acne ". A random sample of 51 consecutive military cases of this disease were therefore studied during August 1945 at Guam.

The malady which Sulzberger and his colleagues call " tropical acne ", was definitely a true acne, and not furunculosis, folliculitis or any other form of pyodermia. The lesions were blackheads, papules, pustules and cysts ; the local process was identical with acne vulgaris of temperate climates, but differed from that disease in certain respects, *viz.* :—the average age of onset in the series studied was 22 years, although in twenty-four of the cases no history was obtained of adolescent acne ; the lesions tended to involve many areas such as the forearms, abdomen, buttocks and thighs which usually are free in typical acne vulgaris. Often one or more of these areas was severely involved while the face was not affected ; nodules, cysts and scars were much more uniformly present and were more severe than would have been expected in a similar random group of cases of acne vulgaris.

So long as the patients remained in the tropics, orthodox methods of treatment either failed or produced only partial or temporary benefit. Rapid improvement usually occurred when the patient reached a cool dry climate.

Poor hygienic conditions did not appear to be a primary cause of tropical acne, although poor washing facilities, inadequate accommodation and dietary deficiencies may play some rôle in the aetiology, but heat and humidity, with certain unknown local or constitutional factors, are regarded as sufficient to cause the disease. The authors suggest that the pathological changes may be due to plugging of the sebaceous ducts because of hyperkeratosis caused by sunlight ; this effect is intensified by moisture and maceration ; meanwhile the heat of the environment stimulates the sebaceous glands, which continue to secrete actively, although their ducts are blocked.

Even if severe, previous attacks of acne vulgaris do not necessarily justify the inference that tropical acne will develop when the patient reaches hot and humid climates, for the authors have noted several persons suffering from " active acne " when entering the tropics, whose lesions have either improved

or remained unchanged ; nevertheless they consider that these cases are more likely to develop tropical acne than the normal individual, and recommend that persons with widespread acne with cyst formation, or with excessively oily skins or with any other signs of hyperactivity or dysfunction of the sebaceous glandular system, should not be sent to the tropics. These criteria are not infallible, and they know of none whereby persons prone to tropical acne can be recognized with certainty before they are posted abroad.

[The experience of the British Army was somewhat similar to that noted by Sulzberger and his colleagues ; but we recognized one factor which they do not stress. Acne tends to be particularly troublesome in the tropics among troops who have to wear webbing equipment or are subjected to the friction and rubbing of clothing, particularly if a large proportion of the men have suffered from acne vulgaris in temperate climates. Whether or not a quiescent acne would flare into activity in the tropics often depended very much on the duties which the individual had to perform.] *R. M. B. MacKenna.*

CORMIA, F. E. & ALSEVER, W. D. **Uses and Abuses of Penicillin in Dermatology.** *Arch. Dermat. & Syph.* 1946, Aug., v. 54, No. 2, 136–49.

The theory on which much of this paper is based can be summarized by saying that various strains of staphylococci have varying degrees of resistance to penicillin. Therefore it is not rational to prescribe a standard ointment or cream containing, say, 500 units of penicillin in each gramme for every case in which the cutaneous lesions are apparently due to invasion of the skin by these bacteria ; it is better to ascertain the penicillin resistance of the staphylococci isolated from each case by bacteriological methods, and then to prescribe a penicillin ointment of suitable potency.

A group of 75 patients suffering from treatment-resistant pyoderma (impetigo, ecthyma, sycosis, folliculitis, etc.) was selected for study. Usually their lesions were due to a pigmented, coagulase-positive, haemolytic *Staphylococcus aureus*. The organisms isolated from each case were smeared across a blood agar plate ; disks of filter paper, 6 mm. in diameter, were saturated with varying dilutions of penicillin solution and placed on the plates. A Florey strain of *Staph. aureus* which was uniformly sensitive to disks saturated with a penicillin solution containing 1–2 units of penicillin per cc. was used as a control. These studies showed that in 75 per cent. of the cases, the causal organisms were insensitive to penicillin in a strength of 2 units per cc. and it became evident that in the group of 75 patients only 64 per cent. were likely to be cured by local therapy when the concentrations of penicillin normally used for this purpose were applied.

Varying concentrations of penicillin were used in penicillin ointment in a group of 15 patients in whom the organisms were sensitive to penicillin in concentrations ranging from 4 to 2,000 units per cc. The ointment base was made of 15 per cent. petrolatum emulsified with 15 per cent. Lanette Wax in water. Three patients with chronic impetigo and one with ecthyma whose organisms were sensitive to penicillin in a strength of 4 units per cc. were cured within 10 days by the use of ointment containing 3,000 units per gramme. Three other cases suffered from staphylococcal infections of the skin, the bacteria being sensitive to penicillin in dilutions of 6–10 units per cubic centimeter :—

" The first had an infectious eczematoid dermatitis of the face of eight months' duration. The process was arrested temporarily during three previous hospitalizations with the use of penicillin ointment (500 units per gram), but there was invariably a relapse from seven to fourteen days after therapy was stopped. Culture revealed a coagulase-positive hemolytic *Staph. albus*, sensitive to

penicillin in a strength of 6 units per cubic centimeter. Treatment was instituted with an ointment containing 5,000 units of penicillin per gram, and complete involution occurred after two weeks of therapy. Treatment was then continued for another two weeks to insure permanency of results."

"The last patient in this group had been hospitalized almost continuously for the preceding eight months because of a treatment-resistant sycosis vulgaris. Three million units of penicillin had been administered intramuscularly in eight separate courses, and penicillin ointment had been used for three weeks without affecting any improvement. On culture a coagulase-positive hemolytic *Staph. aureus* was found, with sensitivity to penicillin in a strength of 20 units per cubic centimeter. Penicillin ointment in a strength of 100,000 units per gram was used, and cure was accomplished with three days of therapy. In all, treatment was continued for five days, and there was not the slightest evidence of local irritation. The patient was followed for another month, but relapse did not occur."

Despite these successes, failures were recorded in other cases, although in some instances penicillin ointment containing 3,330 units per gramme was used, and in one case the ointment was loaded with 10,000 units per gramme. Therefore the authors conclude that even massive penicillin therapy may be inadequate, and may lead to the development of increased penicillin resistance.

Cormia and Alsever consider that the only obvious indication for the administration of penicillin intramuscularly is in cases of severe, widespread infectious eczematoid dermatitis.

In the treatment of 10 patients suffering from pyoderma, sulphadiazine (5 per cent.) was added to penicillin ointment of standard strength (500 units per gramme); the results were disappointing. However, benefit did seem to accrue in four patients who received two courses of penicillin locally with an intervening oral course of sulphadiazine.

As the study progressed, the filter paper method of bacteriological assessment was given up and the authors adopted, as more accurate, a method of assessment devised by COOKE [*Bulletin of Hygiene*, 1945 v. 20, 497] in which penicillin solutions of varying strength and measured amount (0·1 cc.) are applied evenly to the agar and dried quickly in the refrigerator; the organisms to be tested are then streaked across the area and the inhibition of growth, or its absence, noted as usual.

As a result of their studies they suggest the following scheme of correlation between the strength of penicillin ointment and the sensitivity of the organisms concerned :—

Strength of penicillin ointment to be used in treatment according to sensitivity of organisms to penicillin.

Sensitivity of Organism		Strength of penicillin units per gramme of ointment
Filter paper method, units per cc.	Cooke method, units per 0·1 cc.	
1	0·03 to 0·06	500
2	0·1	1,000
4	0·15	3,000
6	0·2	5,000
8	0·25	7,500
10 to 20	0·3 to 0·6	10,000 to 25,000 (?)
20 to 30	0·6 to 1	25,000 to 50,000 (?)
30 to 50	1 to 2	100,000 (?)

R. M. B. MacKenna.

JACKSON, W. P. U. **Plant Dermatitis in the Bahamas.** [Memoranda.] *Brit. Med. J.* 1946, Aug. 31, 298.

The shrub *Metopium toxiferum* (family Spondiaceae), known as poison wood or poison bark, contains a toxin at least as powerful as that of *Rhus toxicodendron.* It flourishes throughout the open spaces of the Bahamas and Southern Florida. The toxin is present in the " milk " beneath the bark, in the leaves and in the fruit, and tends to ooze to the surface of bark and leaves, producing black stains.

After contact of the skin with this toxic resin, there are usually no symptoms for a few days, but thereafter small papules are formed, which rapidly become vesicular ; there is itching and surrounding erythema, and the lesions usually ulcerate. The rash may spread to distant parts of the body, probably as a result of a general allergic reaction of systemic dissemination, since blister fluid is not toxic and finger-spread is therefore not likely. The whole process may continue for several weeks. Sometimes the rash is more urticarial, with gross erythema and oedema. One drop of the fluid may produce a widespread rash.

Charles Wilcocks.

SAXTON, W. J., HATCHER, F. & DERRICK, E. H. **Chromoblastomycosis, with Reports of Two Cases occurring in Queensland.** *Med. J. Australia.* 1946, May 18, v. 1, No. 20, 695–7, 6 figs. on 1 pl.

Chromomycosis, or mossy foot, although found in many parts of the world, is not common in any country. The authors describe two cases, the first to be reported in Australia. In both cases the lesion was a small, painless, non-inflammatory plaque about 15 mm. in diameter, with a flattened surface elevated a few millimetres above the level of the surrounding healthy skin. On one patient the lesion, which had evolved from a small wart-like papule, was of 7 years' duration and on the other a matter of only 5 months. Topical treatment proved ineffective and excision was practised in both cases. Histologically the lesion was a cutaneous granuloma composed of histiocytes and plasma cells with polymorphonuclear and mononuclear leucocytes, numerous giant cells of the foreign-body type, and many very conspicuous eosinophil cells. The fibroblastic reaction was very slight but there were marked epidermal proliferations which, in neglected cases, give rise to the papillomatous character of the lesions. The fungal parasite was seen singly or in little groups scattered throughout the tissue, often within giant cells or forming the central object of a little " abscess " of polymorphonuclear cells. The fungal cells were round or oval, from 5 μ to 14 μ in diameter, averaging 8 to 10 μ ; they were of a brown colour, thick-walled and, in some cases, septate. Unfortunately, the tissues were fixed in formol before the diagnosis was made, so the identification of the fungus by culture was not possible.

J. T. Duncan.

PARÁ, M. **Histoplasmosis in Brazil.** *Amer. J. Trop. Med.* 1946, May, v. 26, No. 3, 273–92, 17 figs. [36 refs.]

The author, who is a member of the National Yellow Fever Service of Brazil, diagnosed five cases of histoplasmosis in the routine histological examination of 169,808 viscerotomy specimens collected in connexion with the yellow fever survey, during the period 1939 to 1945. As the diagnoses were made some time after death, confirmation by cultivation of the fungus was not possible, and the case histories obtained were incomplete. All five patients were children, 2 boys and 3 girls, between the ages of 19 months and 9 years. The cases occurred in the following districts : Recreio, Minas Gerais ; Xiririca, São Paulo ; João Alfredo, Pernambuco ; and Itapé, Bahia (2 cases). Four were diagnosed

during the current routine examination for yellow fever, and the remaining case on a re-examination of specimens taken in Itapé in 1939, in consequence of the discovery of a case in the district in 1944. One of the children, a boy aged 19 months from Xiririca, São Paulo, was known to have played with three emaciated dogs, and a subsequent examination of these animals showed one of them to be suffering from histoplasmosis, the diagnosis being confirmed by isolation of *H. capsulatum* from the animal's lung. There was no means of knowing whether the child or the dog was infected first. This is the fourth published case of naturally acquired histoplasmosis in the dog, and the first in which the animal was associated in any way with a known human case.

J. T. Duncan.

CARINI, A. & SPRINGER, L. Caso humano de pequeno cisto subcutâneo cheio de esporos de um cogumelo não identificado. [**A Subcutaneous Cyst in Man containing Elements of an Unidentified Fungus.**] Reprinted from *Arquivos de Biologia.* 1944, Mar.–Apr., v. 28, No. 260, 4 pp., 5 figs.

TROPICAL OPHTHALMOLOGY.

A REVIEW OF RECENT ARTICLES. XLVII.*

The eye conditions in the Aborigines in the Northern Territory of Australia are discussed by SCHNEIDER[1]. Collections of dark brown pigment in the bulbar and tarsal conjunctivae of adults are commonly found. They tend to be localized to the larger accumulations in the areas normally exposed when the eyes are open. The caruncle and the plica semilunaris sometimes share in this lavish distribution of pigment. Infants and children are not affected and so these collections of pigment must be regarded as having been acquired. Occasionally a complete ring of pigment surrounds the cornea, usually but not invariably separated from the limbus by a space of about one millimetre. The colour of the iris is always dark, varying from chocolate-brown to almost black. The fundi are almost invariably of a uniform slaty-pink colour and choroidal vessels cannot be seen. By contrast, the optic nerve head is apparently, but not actually, paler than in European eyes. The visual acuity as a whole approximates that of Europeans but in the field of vision an aboriginal leaves us far behind in perceptive powers. These are not due to any advantage in visual acuity but are rather the result of training and careful observation. Errors of refraction are due to hypermetropia and hypermetropic astigmatism. In eyes free from corneal disease, myopia is unknown. In diseases of the eyelids, chalazion is not uncommon, but blepharitis and hordeolum are rare. Chronic dacryocystitis in association with old, active, severe trachoma is not uncommon. In diseases of the conjunctiva, pterygium is rare and is considerably less common than among the white population residing in the Northern Territory. Mucopurulent conjunctivitis is prevalent in children, and the organisms found most commonly are Koch-Weeks bacilli and pneumococci. Arcus senilis is not uncommon in elderly people, and owing to their outdoor life in thick high grass and bush, the natives in the northern areas occasionally present corneal abrasions. The aborigines appear to be free from disorders of the uveal tract, and gonococcal eye disease is unknown. The incidence of yaws is markedly high, and if it is assumed that infection by *Treponema pertenue* immunizes a people against infection by *Treponema pallidum*, then syphilis is

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* For the 46th of this Series see Vol. 42, pp. 1030–1037.

[1]SCHNEIDER, M. A Sociological Study of the Aborigines in the Northern Territory and their Eye Diseases. *Med. J. Australia.* 1946, Jan. 26, v. 1, No. 4, 99–104.

not likely to be encountered in the aborigines ; and it is probable that cases of interstitial keratitis, optic atrophy and central retinitis may be regarded as unusual manifestations of yaws.

Trachoma, as may be expected in a people living in close mutual contact, without any conception of personal cleanliness and hygiene, has a markedly high incidence. Among 574 subjects, 520 (90 per cent.) presented evidence of trachoma in one of its stages. The most remarkable feature of the disease is the extraordinary influence that climate bears on its severity. In the southern areas of the Northern Territory, which are rarely free from wind and dust, the disease is severe ; in the northern areas, which are comparatively free from these factors, the disease is mild and complications are rare. The incidence rate is almost identical in the two areas. The chief vehicle in the transmission of the organism of trachoma in the aboriginal is almost certainly the human finger. It is a common experience to observe a woman wiping the secretion from her eyes with her fingers, and a moment later repeating the manipulation on her child's eyes, wiping off any excess on some part of her body. Flies are prevalent in the southern areas but play a minor rôle, if any, in the dissemination of the disease. The severe variety of trachoma conforms to the classical description, in that it commences in early childhood and terminates only at death, producing more suffering than any other disease, and is accompanied by progressive impairment of vision and not infrequently by blindness. In the mild variety, very few patients complained even of discomfort, yet on examination pannus with follicles and epithelial hyperplasia was present in all cases.

Blindness in Nigeria.—In September 1944, a census of all blind persons was taken by STOCK[2], in Yerma, the chief town of Bornu Province, Northern Province in order to ascertain the percentage of blind persons in the community, and as far as possible to determine the cause of this blindness. The figures recorded are probably representative of other urban centres in Northern Nigeria which experience a similar climate and in which the same diseases are prevalent. Yerwa is a town of approximately 35,000 persons, situated in the north-eastern corner of Nigeria and at the southern edge of the Sahara Desert. The climate is intensely dry during the greater part of the year, and dust storms are very common. The greater part of the average annual rainfall of 25 inches falls in the months of July to September, with slight showers between April and June and in early October. The figures which record the total number of blind persons were computed by the local Native Authority, and may be taken to be fairly accurate but they are probably slightly below the correct figure. Rather more than 50 per cent. of all the blind persons in the town were examined ; a representative number of persons of both sexes and of all age groups, except young children was included. It was found that the incidence of blindness among the community is approximately 1·4 per cent., but it is certain that this is an under-estimate owing to concealment of cases in young persons ; the correct figure is probably between 2 and 2·5 per cent. In any case, the recorded percentage of blindness in these natives of West Africa is considerably lower than that (5 per cent.) given by ROSSET BERDEZ for South African natives.

An analysis of blindness in infancy (under 5 years of age) revealed that conjunctivitis and smallpox are the predominant causes. In view of the very septic conditions which prevail at childbirth and the very high incidence of venereal disease in this part of Nigeria, the author is of opinion that these cases of conjunctivitis should be classed as ophthalmia neonatorum, bringing the percentage of blindness due to this cause to 11·4 per cent.

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[2]STOCK, F. E. Blindness in an Urban Centre in Nigeria. *Brit. Med. J.* 1946, Apr. 6, 525–6.

In the causation of blindness in all age groups, conjunctivitis and its complications were responsible for 103 out of 254 cases, smallpox for 103, cataract for 26, and glaucoma for 10 cases. Many cases of severe conjunctivitis showed evidence of keratomalacia, a condition which is also very prevalent. [No mention is made of trachoma as a cause of blindness.]

It is believed that at least 80 per cent. of the cases of blindness could be prevented by widespread compulsory vaccination, by the provision of a more suitable dietary containing vitamin A, and by extending medical facilities in order to ensure that all cases of conjunctivitis should receive adequate treatment.

Trachoma.—During the past eighteen years WILSON[3] has had a unique opportunity of studying trachoma from many varied angles, and in this article he reviews certain personal observations made during that period. In discussing the aetiology of the disease he states no one has yet succeeded in isolating the infective agent of trachoma in pure culture, but there is no doubt that it is a specific infection. He rejects the theories of a lymphatic dyscrasia, adenoidism, nutritional deficiency or similar concepts, and does not consider these factors to be predisposing causes.

He regards the evidence in favour of a rickettsia as a cause of trachoma to be far from convincing. *Bacterium granulosis* he regards as the cause, not of trachoma, but of a simple form of chronic granular conjunctivitis. He is convinced that typical intracellular inclusion bodies are invariably present in the incipient stages of trachoma and also that, if they are not demonstrable in the early stages of any disease under examination, the disease present is not trachoma. In this connexion, it is to be observed that inclusions may be found in the trachomatous conjunctival epithelium some appreciable time before characteristic clinical signs appear in the tarsal conjunctiva; but as the disease progresses they rapidly decrease in number and the chances of finding them are correspondingly diminished after the first three months from onset. Therefore, while the demonstration of the inclusions in the earliest stages of any doubtful case is of the utmost significance, their absence in the later stages is, from the diagnostic point of view, of little importance. There is no doubt now that trachoma is caused by a filterable virus and that the specific infectious agent is demonstrable as an intracellular inclusion body identical with the chlamydozoa originally described by PROWAZEK and HALBERSTAEDTER.

Wilson discusses the problem of animal experimentation and is of opinion that the ideal experimental animal for trachoma has yet to be found. It would appear that the Sudanese monkey (grivet or *Lasiopyga griseoviridis*) is the most susceptible of all the monkeys and apes. When such animals are directly inoculated with trachoma material, mild conjunctival congestion and follicles begin to appear in the upper fornices after a fortnight or so, and sometimes quite a considerable crop of follicles may appear; but these gradually retrogress and may perhaps entirely disappear after several months. Appearance of T.1 follicles on the tarsi has never been observed, nor does scarring of the conjunctiva or vascularization of the cornea ever take place. Furthermore, intracellular inclusions are not found. The position is even more complicated since monkeys may also suffer from a form of spontaneous folliculosis of varying severity which is indistinguishable clinically from the disease produced by the inoculation of trachomatous material; and in this disease also intracellular inclusions are not found. BLAND[4] has shown that trachoma can definitely be

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[3]WILSON, R. P. Trachoma. A Selection of Personal Observations and Experiences. *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 13–37. [16 refs.]

[4]BLAND, J. O. W. The Aetiology of Trachoma. A Critical Review of Present Knowledge. *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 89–106. [58 refs.]

passed from monkey to monkey, through man, producing all the pathognomonic lesions of the disease, and back again to the monkey; spontaneous folliculosis is not transmissible in this way. When simian trachoma is transferred to the human conjunctiva, typical lesions of the human disease are developed, and Prowazek-Halberstaedter inclusion bodies are present in abundance. Thus the virus of trachoma, although not demonstrable in the conjunctival epithelium of the monkey, nevertheless rapidly develops and produces typical colonies after transfer to the human conjunctiva.

In Egypt, trachoma is not, in the true sense of the word, epidemic; it is pandemic. It makes its appearance at all times of the year and may be found in 100 per cent. of the village population and only to a slightly less degree in the big towns. The disease develops in children before they are a year old, and is invariably preceded by a Koch-Weeks conjunctivitis. The usual interval which elapses between the onset of an acute muco-purulent conjunctivitis and the first definite clinical signs of trachoma varies from 6–12 weeks. The virus of trachoma is carried across from eye to eye by flies at the same time as the acute bacterial infection. Fingers are not important in dissemination [but compare SCHNEIDER, above]. The influence of climate, temperature, altitude, geographical distribution, dust, racial predisposition, and the like, is in no way causal but merely concomitant. Bad hygienic conditions are also only predisposing factors. If the fly scourge in Egypt could be controlled effectively there would be a spectacular and immediate radical change in the incidence of trachoma and acute ophthalmia alike.

Egyptians suffering from trachoma react very differently from Europeans, for the tendency towards spontaneous cure is much more evident among the former. This is due probably to a relative degree of inherited immunity rather than to attenuation of virus. The contagiousness of trachoma is not nearly so high as it has always been considered to be, except perhaps in the earliest stages when there is a certain amount of active secretion from the eyes. The superposition of an acute or chronic bacterial infection, which thus produces an increased quantity of infective material available for the dissemination of the disease, is the factor which most enhances the contagiousness of the disease.

Formerly T.1 follicles were regarded as the principal pathognomonic sign of trachoma, but more recent research has proved that there are certain other signs just as pathognomonic. These are not to be looked for in the palpebral conjunctiva, but on the globe. From the 5th–7th day after infection, during the incipient stage of the disease, a narrow band of raised limbal oedema, skirting the upper half of the cornea and tailing away in the lower half, gradually makes its appearance. It is due to commencing infection of this part of the eye, for examination with a corneal loupe or a slit lamp inspection of the limbus shows all the characteristic signs of superficial corneal invasion, *i.e.* engorgement of limbal vessels, superficial limbal corneal infiltrations, extension of the end capillary loops into the cornea, etc. Very soon after the cornea begins to show definite signs of invasion, this limbal band of oedema becomes obscured as the mild oedema becomes more generalized.

In the differential diagnosis of follicular conjunctivitis and chronic conjunctivitis from trachoma, the absence of invasive signs in the limbus is a certain proof that the disease is not trachoma; and if the condition is of short duration an epithelial scraping is of considerable value, for inclusions can almost certainly be found if the disease is trachomatous. If one of these diseases is superimposed upon an old trachoma, then it may be impossible to decide upon the true condition. Under the circumstances it is always rational to treat the element of mixed infection first.

Prophylaxis against trachoma in Egypt has been disappointing, and so far has only been of benefit in delaying the onset of the disease a little; for many

years to come little amelioration can be expected unless some epoch-making medicament is discovered which will effectively control and prevent the disease. The sulphonamides (sulphapyridine, sulphathiazole and sulphadiazine) have proved disappointing in the treatment of trachoma ; but, on the other hand, these drugs have proved so highly and so rapidly effective in the treatment of the acute ophthalmias that, provided they are given early, it is within the bounds of possibility that they may be capable not only of controlling the regular seasonal epidemics of these acute diseases, but also, indirectly, of reducing very considerably the incidence of trachoma at the same time.

The aetiology of trachoma is also discussed by Bland. At present, only two theories are generally accepted, the virus theory and the rickettsial theory. The conflict is indeed one of nomenclature rather than of fact and largely depends upon what we choose to call a rickettsia, and what we call a virus. The evidence so far accumulated leads to the conclusion that trachoma is a specific infectious disease, not caused by any culturable bacterium, but due to a filter-passing agent which is identical with the elementary and initial forms found in the inclusion bodies (Prowazek-Halberstaedter inclusions), and bearing a close natural relationship to the viruses of inclusion conjunctivitis, lymphogranuloma inguinale and psittacosis. There is no evidence that this agent possesses an arthropod host, nor that the louse is the vector of the disease, though the agent may survive for some days in the body of the louse. Bland considers that these four agents stand in an intermediate position between the rickettsiae and the large viruses, and may possibly form a biological link between them. For the present he would prefer to group them with the viruses but to give them a distinctive position as the " basophilic viruses ", on account of the blue staining of their initial bodies and of the matrix of their inclusions which distinguish them from the larger typical viruses, which do not possess blue initial bodies and whose inclusions are acidophilic.

The bacteriological and clinical observations on the treatment of the acute ophthalmias of Egypt (*i.e.* those due to the Koch-Weeks bacillus and the gonococcus) with sulphonamides and penicillin are discussed by Bland and Wilson.[5] The untreated case of acute ophthalmia discharges living and virulent organisms from the eyes for at least 6 days, during the whole of which period the patient is a source of infection for others. By sulphonamide treatment, this period can be reduced to perhaps 1 or 2 days, for any case will be rendered non-infective 12 hours after the first administration of the drug ; and after 2 doses of the drug in every case the conjunctival discharge will be bacteriologically negative for at least the whole of the day following the 2 doses, even in the cases that relapse.

The authors found that sulphapyridine, sulphathiazole and sulphadiazine are all efficacious, but as sulphadiazine is the least toxic it is likely to be the drug of choice. The calculation of dosage according to body weight is not always convenient or practical, and they suggest a dosage based on age, the following figures serving as a guide [one tablet contains half a gm.] :—

0–3 months	$\frac{1}{4}$	tablet	twice	daily.	
3–6 ,,	$\frac{1}{3}$	,,	,,	,,	
6–12 ,,	$\frac{2}{5}$	,,	,,	,,	
1 year	$\frac{1}{2}$	,,	,,	,,	
3 years	$\frac{3}{4}$	,,	,,	,,	
6 ,,	1	,,	,,	,,	
10 ,,	$1\frac{1}{2}$	,,	,,	,,	
20 ,,	$2\frac{1}{2}$	,,	,,	,,	

[5]Bland, J. O. W. & Wilson, R. P. Bacteriological and Clinical Observations on the Treatment of the Acute Ophthalmias of Egypt with Sulphonamides and Penicillin. *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 49–71. [10 refs.]

The best way in which to give the drug is to suspend the dose in 2–3 cc. of milk and drop it into the child's mouth with an eye dropper. An alternative is to make the dose up into a paste with milk or water and to insert it into the child's mouth on the end of a finger. The authors consider that penicillin is quite unsuitable for the treatment of the acute ophthalmias, certainly in Egypt, if administered intramuscularly in the form of eye drops, for it would impose an enormous strain on the nursing staff; and, furthermore, the Koch-Weeks bacillus is not sensitive to penicillin. Acute ophthalmia is the cause of 80 per cent. of the blindness of Egypt and leaves behind, in addition, a multitude of more or less serious ocular defects. The increased ocular secretion caused by the disease promotes also the spread of trachoma, that second universal plague of Egypt, itself a fertile source of damaged sight. No treatment which requires hospitalization or even attendance at hospital, no treatment which cannot be taken to the villages, where the great majority of the people live, and there simply and easily administered, can hope to do more than scratch the surface of this problem. The treatment with sulphonamides completely satisfies the conditions. It is simple, safe, effective and could be given by unqualified persons with a minimum of training; it provides the necessary solution, not only for Egypt but also for other tropical countries where similar conditions of life and disease prevail.

Phlyctenular Ophthalmia in Adults is reviewed by SHAH.[6] A series of 484 cases were examined in the eye out-patient department of St. Mary's Hospital, Lahore. Of these patients, 138 (28·9 per cent.) were over 20 years of age. In this series males comprised 66·6 per cent. and females 33·3 per cent. This is at variance with the widely-held view that the disease is one of childhood and early adolescence and that girls are more affected than boys. Apart from these variations in the age and sex distribution there does not appear to be any other significant difference in the clinical signs and symptoms between the disease in adults and in children. The author's findings agree with those of WANG in Shanghai.

Epidemic Kerato-conjunctivitis associated with Skin Lesions is discussed by O'DONOVAN and MICHAELSON[7]; 33 cases were seen. In all cases the scalp or face was affected and there was a strong tendency for the skin and eye lesions to be ipsilateral. In the majority the onset of the skin lesion preceded that of the ocular lesion by a definite, but short, interval of time. It would appear that lesions of the face and scalp, especially those of a seborrhoeic nature, may be conducive in some patients to the development of a kerato-conjunctivitis identical with the virus kerato-conjunctivitis occurring in epidemic form. The kerato-conjunctivitis in patients with lesions of the skin is rather more intense as indicated by the greater number with bilateral involvement, adenopathy and deep corneal spread, as well as by the slightly longer duration. It is possible that the virus of epidemic kerato-conjunctivitis can possess dermotropic qualities as do the viruses of herpes simplex and herpes zoster. In this case the lesions of the skin may be considered to be analogous to those found in the skin of patients suffering from herpes simplex and herpes zoster.

Penicillin in the Treatment of Common External Eye Infections in out-patients is described by MINTON.[8] The clinical results in blepharitis, conjunctivitis, lachrymal sac infections and industrial eye injuries are excellent. Penicillin

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[6]SHAH, M. A. A Note on Phlyctenular Ophthalmia in Adults. *Indian Med. Gaz.* 1946, Jan., v. 81, No. 1, 20–22.

[7]O'DONOVAN, W. J. & MICHAELSON, I. C. Epidemic Kerato-Conjunctivitis associated with Skin Lesions. *Brit. J. Ophthalm.* 1946, Apr., v. 30, No. 4, 193–204, 12 figs. (including 3 in colour & 5 others on pls.).

[8]MINTON, J. Penicillin in Treatment of Common External Eye Infections. *Brit. Med. J.* 1946, Sept. 7, 324–6, 1 graph. [19 refs.]

is prescribed in the form of eye drops and ointment. He advocates the following method of preparation of penicillin eye drops. These contain 1,000 or 2,000 units per ml. in sterilized distilled water. Twenty ml. of distilled water are poured into bottles fitted with bakelite caps and rubber washers and then autoclaved at 10 lb. (4·5 kgm.) pressure for half an hour. When penicillin drops are required two or four tablets of sodium penicillin (B. & W.) according to the strength required, are added to each 20 ml. The tablets supplied are of three different strengths—9,500, 10,000 and 10,500 units per tablet. The method of preparation of the ointment is as follows :—it is prepared in the strength of 400 units per gramme ; 200,000 units of penicillin in powder form are rubbed in a sterile mortar with a little sterile liquid paraffin ; 500 gm. of eucerin L.M. base (previously sterilized by heating in a sterotherm at 150°C.) are gradually incorporated. The ointment is transferred to sterile jars by means of a sterile spatula.

A biological assay of the penicillin eye-drops proved that the content of penicillin when the eye-drops were kept at room temperature, and the bottles frequently opened and exposed to air, was 80 per cent. of its original potency at the end of fourteen days. The penicillin ointment was prepared without water, and its stability probably lasts longer than six weeks. The drops and ointment have an ideal *p*H value for ophthalmic use and are therefore not irritating to the eyes. A number of adults, after using penicillin drops or ointment, showed allergic skin reactions of the lids and the skin of the face. These cleared up in a few days when penicillin was stopped.

The use of Penicillin in Ophthalmology is also reviewed by PITTAR[9]. He points out that its application is strictly limited and its main use is only in superficial infections of the eye by local application, *i.e.* for conjunctivitis, corneal ulceration, and blepharitis. It may also be found to have a great field in local prophylaxis, in preventing infection following corneal injuries in miners and industrial workers generally ; and it may practically abolish infection following intraocular operations. For local application he advocates drops with a concentration of 2,500 units per cc. ; frequency of application is essential. Clarification is needed regarding the use of penicillin in ointment form and in lamellae. Injections of penicillin into the anterior chamber and vitreous are probably only justified in desperate cases. For intraocular infection there seems to be a considerable field for the use of subconjunctival injection and there appears to be fairly good tolerance to daily injections of 500 units in 0·5 cc. of water. The various methods suggested such as corneal bath, ionization, injections into the anterior chamber and vitreous all have special disadvantages.

[See also abstracts by DARIUS and JOHNSTONE in this section below.]

Malaria.—The ocular complications of malaria are reviewed by GRANT[10]. Opinions on the frequency of ocular complications in malaria have been divergent, depending considerably on variations in the definition of the term " complication " used by the different observers, as well as on the type of disease and thoroughness of examination. In some instances " complication " has been used to signify little more than coincidence, whilst in others the term has been restricted to those ocular lesions which could be identified as local effects of the plasmodia.

On inspection of the figures available for the incidence of ocular complications from the standpoint of changes in the eyes which may occur as the result of the development of acute or masked malaria in otherwise healthy subjects, it

[9]PITTAR, C. A. The Use of Penicillin in Ophthalmology. *New Zealand Med. J.* 1946, June, v. 45, No. 247, 231–5.

[10]GRANT, W. M. Ocular Complications of Malaria. *Arch. Ophthalmology.* 1946, Jan., v. 35, No. 1, 48–54. [57 refs.]

appears that the values of 10 per cent. (PONCET, 1878) and 20 per cent. (SALZIR, 1890), which applied to a selection of chronically and seriously ill patients, might be excessive, whereas the opinion of ELLIOT (1920) that ocular manifestations were rare in India, was influenced by a reluctance to consider as a complication of malaria any ocular disease unless it could be demonstrated to be caused directly by the plasmodium.

DEDIMOS (1932), working in Macedonia, stated that the incidence of ocular complication was between 10 and 20 per cent. CARLOTTI (1918), in Greece, gave the figure of nearly 10 per cent. FIALHO (1927), in Brazil, reported an incidence of 15 to 20 per cent. TOULANT (1938), in Algiers, concluded that ocular complications were relatively infrequent. Accounts of experience with ocular complications in World War II by TALBOT (1943) and ROBERTSON (1944) give the impression that ocular manifestations are relatively frequent, although no actual figures are given.

Most observers agree that dendritic or herpetic keratitis is the most frequent ocular complication of malaria. Much less often interstitial keratitis has been noted. The keratitis usually occurs in one eye only, and its onset is marked by discomfort, photophobia and lachrymation. The cornea is hyperaesthetic, and the lesion stains with fluorescein in the typical dendritic pattern characteristic of the herpes simplex virus in the corneal epithelium. Interstitial keratitis is associated with clouding of the stroma but without vascularization, and severe forms have been described as keratitis profunda and disciform keratitis.

Haemorrhages in the ocular fundus have been noted by many observers. Small multiple retinal haemorrhages are usually located in the periphery of the retina, and visual disturbances are, therefore, not observed ; the haemorrhages are readily re-absorbed and rarely lead to retinitis proliferans. Large retinal haemorrhages are usually central, and temporarily destroy central vision. They are much less common than the small haemorrhages, and are limited to malaria with severe anaemia.

The ocular neurological lesions of most significance so far as they affect vision are those of the retina and optic nerve, or of the pathways of the brain. The common manifestations are scotomas or loss of vision lasting from a few minutes to several hours, followed by severe headache and persistently diminished visual acuity. Dizziness, orbital pain and tenderness with photophobia occur. Changes in the optic nerve and in the fundus sufficient to account for the amblyopia and amaurosis have been described in some cases, while cerebral lesions have been considered responsible for the disturbances in others. Those lesions resulting in loss of vision which are evident ophthalmoscopically, are optic neuritis or atrophy of the optic nerve, and degenerative or haemorrhagic lesions of the retina and choroid. Opacities in the vitreous, iritis and cyclitis are rarely reported except possibly for a mild iritis which occasionally accompanies herpetic keratitis. Cataract is also a rare complication. In the consideration of the usual disturbance in malaria the possibility of toxic effects of quinine in therapeutic use must be ruled out. The changes associated with toxic amblyopia or amaurosis due to quinine are sufficiently typical to be distinguished from malarial manifestations. Amblyopia and blindness due either to minute or massive doses of quinine are rare. At present the mode of toxic action of quinine on the retina is controversial and centres on the question whether the effects of the drug are a result of ischaemia due to alterations produced in the vessels, or of a direct toxic action on the neural elements themselves, but recent opinion seems to favour a direct action of quinine on the neural elements of the retina as a primary factor.

Relatively infrequently, transient ocular motor disturbances, seldom with permanent paralysis, have been ascribed to malaria. Of these, paresis of the abducens nerve and facial paralysis with lagophthalmos have been reported.

Treatment of these ocular disturbances with anti-malarial drugs has in general been successful, except in the case of herpetic keratitis, caused by a virus, for which specific chemotherapy is lacking.

The ocular manifestations in malaria are also discussed by BHATTACHARJYA and GUHA[11]. In the conjunctiva, marked pallor may be noted in malarial haemolytic anaemia, and icteroid discoloration in the bilious remittent type. In the cornea, herpes simplex keratitis, varying from mild forms to dendritic ulcers, is found in protracted cases of the illness, more often in the convalescent or in the relapsing stages. In the vitreous and retina, haemorrhages have been noticed in a few cases. In rare cases, thrombosis of the retinal veins, diffuse retinitis, or retinitis proliferans as a sequel to retinal haemorrhages, may be produced, resulting in permanent impairment of vision. In the neurogenic lesions, swelling of the optic disk from 1 to 2 dioptres has been noticed in a number of cases with meningitis and coma. In severe cases, papilloedema, with exudates and haemorrhages, was seen, with dilated pupils and sluggish or absent light reaction. The malarial amblyopia seen was transitory in nature, without any fundus changes.

Inequality of the pupils, convergent strabismus and nystagmus were also seen. The authors state that quinine amblyopia or amaurosis are not infrequent complications and that retinal and vitreous haemorrhages may occur after large doses of quinine in persons with a haemorrhagic diathesis, and may accompany cutaneous intestinal or urinary haemorrhages.

Scrub Typhus.—The ocular changes amongst 451 patients suffering from scrub typhus are discussed by SCHEIE[12]. The following external changes were seen: conjunctival hyperaemia occurred in 38 per cent. of cases, but disappeared during the second and third weeks of the disease; subconjunctival haemorrhages occurred in 6·6 per cent. The haemorrhages were massive, often covering one-third to one-half of the exposed sclera, and were usually bilateral. Ecchymosis of the eyelids occurred in 1 per cent. of cases. An eschar, typical of the primary lesion of the disease, resulting from a mite bite, was seen, involving the upper lids in 0·5 per cent. of cases. A coarse, irregular, jerky, incoordinate type of nystagmus, occurring only when fixation on an object was attempted, was seen in 0·5 per cent. The following intra-ocular changes were observed. Venous engorgement was the most consistent change in the fundus, and always preceded any of the other intraocular changes; it occurred in 67·2 per cent. of the patients, with onset during the first or second week, frequently progressing until the veins were 2½ times the diameter of the arteries. Accompanying the engorgement were irregularity in calibre of the veins and increased tortuosity of the vessels. Oedema of the optic disk and retina was seen in 36·1 per cent. of cases. These were always both involved and the condition was always bilateral. In some cases, the swelling of the disk was marked and resembled papilloedema. Retinal haemorrhages, usually superficial, were found in 6·6 per cent. of the patients. Fluffy white exudates occurred in 4·9 per cent. These were usually of the "cotton wool" variety, though some resembled gangliform degeneration. The haemorrhages and exudates usually occurred in patients who also had oedema of the retina and disk. Dust-like vitreous opacities, most numerous in the posterior vitreous without further evidence of uveitis, were seen in 4·6 per cent, and definite uveitis in 1·3 per cent. of the patients. The uveitis was usually indolent in character, with slight photophobia and usually without ciliary injection. It was nearly always associated with oedema of the

[11]BHATTACHARJYA, B. P. & GUHA, G. S. Ocular Manifestations in Malaria. *Indian Med. Gaz.* 1946, Feb., v. 81, No. 2, 79–80.

[12]SCHEIE, H. G. Ocular Changes in Scrub Typhus. A Study of 451 Patients. *Bull. U.S. Army Med. Dept.* 1946, Apr., v. 5, No. 4, 423–7, 4 figs.

disk and retina. Visual acuity was undisturbed except in patients with uveitis, who occasionally complained of blurred vision. From the pathological investigations available it would appear that the primary lesion in the eye consists of an inflammation of the highly vascular uveal tract. This might be expected, for one of the characteristic pathological changes of scrub typhus is a vasculitis and perivasculitis of the smaller blood vessels. The incidence of vitreous opacities and bilateral uveitis supports such pathological findings. Even though the clinical picture suggests a retinopathy, the disturbance in the choroid is probably the underlying factor.

Visual Defects in Prisoners-of-War from the Far East are described by Hobbs & Forbes.[13] More than 2,500 men liberated from Japanese prisoner-of-war camps were seen. Defects of vision, recovered or still present, were a prominent feature of the history ; they were classified into three broad groups :—

Group I—defective reading vision only, usually transient ;
Group II—transient blurring of both reading and distance vision ;
Group III—a persistent defect of both reading and distance vision.

Patients in groups I and II mainly presented problems of refraction, and appeared to have had a temporary asthenopia associated with their very debilitated condition before liberation. The visual acuity was within normal limits, and no abnormalies of media, fundi or ocular movements were seen. Group III numbered about 250 persons and comprised a few men with active ocular disease, a number with fully correctible refractive errors, in whose visual fields no scotomata appeared, and a larger group in whom correction with lenses produced little or no improvement, and whose visual fields showed absolute or relative scotomata to white test objects. These cases form a group of 163 cases of partial optic atrophy. The history of 89 per cent. of these 163 cases was of gradual deterioration of vision after the first or second year of captivity, occurring as a general blurring of vision, or as scotomata. The visual loss increases over a period of weeks or months, but a rapid onset was not uncommon and in 11 per cent. the visual loss had developed within twenty-four hours. In many cases the onset was insidious and unrelated to other disease. In regard to refraction, 6 per cent. had visual acuity of less than 6/60 ; 55 per cent. had visual acuity between 6/60 and 6/24 ; 33 per cent., though subnormal, were better than this, and 6 per cent had 6/6 in both eyes.

Pallor of the central halves of the disks was present in most cases. Signs of retinal disturbances were not common and when present were noted between the macula and the disk. Perimetry showed no significant constriction of the peripheral fields in any except the more severely affected cases. Examination of the central fields revealed various types of defects, all of which lay in the centro-caecal area, the most common finding was bilateral absolute central scotomata to 2, 5, 10 or 15/2,000 white, and in such cases the associated loss of visual acuity was correspondingly great. In others, less severely affected, scotomata less dense and less centrally placed appeared, and these often took the form of central and paracentral relative scotomata, with eccentric absolute nuclei, which, in some cases, formed a partial ring and left the fixation area clear. In others there were minute scotomata about the central area, where the 2 mm. white object appeared to " flash on and off."

Group III cases, showing scotomata and partial optic atrophy, were closely related to preceding infection, particularly malaria and dysentery. In the former an increased metabolism and in the latter decreased absorption from the diseased intestine producing a greater lack of those factors which normally prevent the development of this syndrome.

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[13]Hobbs, H. E. & Forbes, F. A. Visual Defects in Prisoners-of-War from the Far East. *Lancet.* 1946, Aug. 3, 149–53, 5 figs. [15 refs.]

The response to treatment was poor. The superiority of animal protein over yeast in prevention and treatment suggests that some factor besides Vitamin B, contained in animal protein, is necessary to prevent the onset of these lesions, and that the lack of protein is a precipitating factor in the disease ; it may well be that the rôle of protein consists in promoting the biosynthesis of vitamins in the intestine.

Nutritional Retrobulbar Neuritis, or nutritional optic neuropathy, is reviewed by MOORE.[14] The aetiology is still far from complete. He is of opinion that it can be excluded from beriberi, though it can be associated with it. The response of the visual condition to autoclaved yeast, and the investigations with thiamin in the prisoner-of-war camps, are impressive evidence against beriberi. In West Africa nearly all cases were either accompanied or preceded by hyporiboflavinosis, but it would not be safe to state that riboflavin deficiency caused the neural condition, for trials with riboflavin have never been sufficiently extended. Lack of riboflavin is no doubt an important causal factor, but other vitamins of the B complex, excluding thiamin, may also share in the cause. The same is partly true for nicotinic acid, though there is more evidence that lack of this vitamin is not directly concerned. Experiences in prisoner-of-war camps appear also to support this view. Treatment has yielded important results, provided it is begun early, the dosage (marmite or brewer's yeast) is high, and it is maintained over a long period.

The clinical features are important, for there are distinct variations and superimposed conditions to be found. Keratitis was rare in Nigeria, but common in Sierra Leone, and apparently in prisoner-of-war camps. The Jamaican cases were fulminating. Beriberi is rare in Nigeria, but common among ships' crews, on a diet of rice and salt fish, from Sierra Leone and Liberia. In Nigeria, retrobulbar neuritis was extremely common, but in certain areas only, or under bad conditions which existed in some schools before the war. It was not seen in those areas where yams or sweet potatoes, or better class cereals, formed the staple carbohydrate. As early as 1933 the author was convinced that manioc was the dietetic cause, for the disease was essentially confined to the manioc-eaters. But in Jamaica manioc was replaced by sugar cane, in Sierra Leone and Spain (civil war conditions) largely by rice, and in China and the Far East solely by rice. Where the rice was unpolished, or, as in prisoner-of-war camps, reinforced with barley and soya bean, there was least evidence of retrobulbar neuritis. A theory based on an exogenous toxin, therefore, can only be upheld if it can be proved to be common to all conditions. There is an obvious alternative explanation—a theory based on an endogenous breakdown. It may be that certain foodstuffs do possess inhibitory toxic factors and also help to accelerate metabolic breakdown (*e.g.* cyanogenetic factors ; alcohol in secondary pellagra).

What has been established is that, under conditions of duress or environmental restrictions, leading to an insufficiency in normal intake of protein (and, to satisfy hunger, a relative over-consumption of energy foodstuffs poor not merely in vitamin B_1 but in all the B vitamins), nutritional retrobulbar neuritis has been common. Other syndromes such as beriberi may, of course, co-exist. Why say that rice can cause only beriberi, when it is poor in all nutrients ? Where there is a gross dietary imbalance, and where cereals of poor quality are almost the sole source of energy, and protein is deficient, then we can be certain that this visual complaint and the associated primary deficiency status must remain. It is not enough to ensure an adequate crop of rice or manioc ; we must also aim at a vast improvement in the variety of carbohydrate foods and in the diet as a whole, and a rise in the economic standards of life.

[14]MOORE, D. F. Nutritional Retrobulbar Neuritis. Nutritional Optic Neuropathy. *Lancet*, 1946, Aug. 17, 246–8, 3 figs. [33 refs.]

Nutritional Optic Neuropathy.—The syndrome of epithelial and nervous lesions complicated by nutritional optic neuropathy is reviewed by WRIGHT.[15] He found defective vision to be a cause of constant complaint and attributed its origin to deficiency of vitamins A and B ; he therefore introduced, in 1927, the " active treatment " with yeast and cod liver oil. In Sierra Leone the dietary is deficient in protein, and hence in sulphur. In 1936 he described the experimental treatment of the syndrome with organic sulphur, using " Contramine " parenterally in some cases, and ichthyol orally in others. Judicious sulphur therapy was of great benefit to the patient and resulted in economy in treatment. Although good clinical results have consistently been obtained by combined sulphur and vitamin-therapy in Sierra Leone, he has seen no record of its use in nutritional optic neuropathy.

The Nature of Starvation Amblyopia in 277 cases in Camp Nakom Paton is analysed by HAZELTON.[16] The aetiology of the disease seems to be bound up with a combination of vitamin deficiencies. The disease resolves itself into two parts : (1) Easy exhaustion of the ciliary muscles, which is cured by the administration of sufficient doses of thiamin, at least 1,000 international units intramuscularly each day for 14 days. Also the symptoms of eye strain (pain in the eyeballs, frontal headaches, excess lachrymation, heaviness of the lids and tired feeling of the eyes which increased after reading) were alleviated by the administration of thiamin ; (2) A condition of degeneration of the cones of the retina which itself is probably brought about by the lack of sufficient photosensitive substance for these receptors. The former is shown (*a*) by lowering of the visual acuity, (*b*) inability to differentiate objects close together, (*c*) interference with colour vision. The latter is postulated from the fact that the colour of print appears to change on reading, from black to green or yellow. Two facts emerge from this :—

1. There is a definite relation between eyestrain and vitamin B_1 deficiency.
2. Sufferers from avitophthalmia are liable to sustain further damage to their cones in bright light ; they should not work in sunlight and should wear dark glasses. Any degeneration of the retina is probably permanent and in early cases every effort should be made to supply a well-balanced diet, failing the elucidation of the specific factor causing the disease. The results of treating cases of nutritional amblyopia of short duration with 10 eggs per day for thirty days were very gratifying. All cases undergoing this treatment showed a distinct improvement.

The Ocular Symptoms and Signs associated with Deficiency of Vitamin B Complex are discussed by FERNANDO, AYUYAO and CRUZ.[17] Their report is based on the observations of 590 adult patients in the Philippines and is the first epidemic of this nature ever reported in that country. The chief complaint was dimness of vision in both eyes, the cause of which was : (1) chronic retrobulbar neuritis, (2) superficial vascularizing keratitis, or (3) both affections co-existing. The patient invariably gave the history of having subsisted for some time on a diet very deficient both in quantity and in quality. They were healthy up to the time of onset of their complaint. Adult males and females were attacked. In females, pregnancy and lactation were predisposing factors. In many patients associated signs and symptoms of vitamin B complex deficiency were observed namely, angular stomatitis, glossitis, numbness around the mouth, hyperaesthesia or hypoaesthesia in the extremities, and other manifestations.

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[15] WRIGHT, E. J. Nutritional Optic Neuropathy. [Correspondence.] *Lancet.* 1946, Sept. 14, 401–2.

[16] HAZELTON, A. R. The Nature of Starvation Amblyopia. *J. Roy. Army Med. Corps.* 1946, Apr., v. 86, No. 4, 171–8.

[17] FERNANDO, A. S., AYUYAO, C. D. & CRUZ, J. N. Ocular Symptoms and Signs associated with Deficiency of Vitamin B Complex. *J. Philippine Med. Ass.* 1946, Mar., v. 22, No. 3, 93–107. [14 refs.]

The retrobulbar neuritis was probably brought about by several known and unknown causes, but mainly by the deficiency of the B_1 factor and especially of the vitamin B complex. Their hypothesis is that the papillo-macular bundle of the optic nerve is susceptible to attack by a toxic substance when there is deficiency of the vitamin B complex and concomitant ariboflavinosis. Likewise, the superficial vascularizing keratitis was brought about by similar mechanism—the deficiency of the B_2 factor of the same vitamin B complex playing a predominant rôle.

The essence of treatment was the administration of a well-balanced and liberal diet, especially rich in those vitamins which are suspected to be insufficient.

The Ocular Manifestations of Avitaminosis in 500 cases of Indian prisoners of war in Singapore are discussed by SARDANA.[18] The great majority were between 30 and 40 years of age. Of these, 159 suffered from vitamin A deficiency, and their chief complaint was difficulty in seeing in the dark. The cornea was insensitive in 81 per cent., and keratinizing epithelial metaplasia was observed in some of them. Not a single case showed xerosis, keratomalacia, or follicular skin lesions. Visual acuity for distance was reduced in all cases, but the media and fundi appeared to be normal. Two hundred and twenty persons were suffering from vitamin B_1 deficiency. The chief ocular complaint was dimness of vision for distance as well as when reading. A burning sensation in the eyes, with discomfort on exposure to sunlight, lachrymation, mild photophobia with some degree of blepharospasm, were present in almost all cases, but the media and fundi were normal. One hundred and twenty-one of these men were suffering from riboflavin deficiency, and complained of hazy vision. Practically all the patients had angular stomatitis, and the conjunctiva showed a streak of vascular infection in the interpalpebral region, extending from the limbus to the outer canthus. The cornea was found clear in all cases except one, which showed sclerosing keratitis with circumcorneal injection. The media and fundi were normal. Eight per cent. of the cases were purely manifestations of tobacco amblyopia. *E. O'G. Kirwan.*

TOLEDO, S. de A. A combate ao tracoma no Estado de São Paulo. "Da organização e funcionamento dos serviços de profilaxia e assistencia médico-sanitária." (Novembro de 1944). [**The Organizations of the Campaign against Trachoma in the State of São Paulo, Brazil.**] *Arquivos de Hig. e Saúde Pública.* São Paulo. 1945, June, v. 10, No. 24, 163–282, 3 figs. (1 folding) & 2 folding maps. [36 refs.] English summary.

DARIUS, D. J. **Penicillin Treatment of Trachoma : Preliminary Report.** *Amer. J. Ophthalm.* 1945, Sept., v. 28, 1007. [Summary taken from *J. Amer. Med. Ass.* 1945, Dec. 1, v. 129, No. 14, 981.]

Darius reports 12 cases of trachoma ranging from type I to the chronic type III treated at the United States Indian Service Hospitals with sodium salt of penicillin in a solution of 500 units to each cubic centimeter of water. Drops of this solution were instilled in the eye every three hours. Improvement was observed under the three-hour schedule but more rapid results were obtained under a half-hour schedule. Loss of photophobia and lacrimation was noticed in twenty-four to forty-eight hours. Improvement in vision was usually evident on the third day, as was paling and thinning of the hyperemic palpebral conjunctiva, with gradual flattening of follicles and granulation tissue. Beginning shrinking and retraction of vessels of the pannus was definite on the fifth to the seventh day, with accompanying slower clearing of

[18] SARDANA, M. N. Ocular Manifestations of Avitaminosis in Adults. *Indian Med. Gaz.* 1946, Apr.–May, v. 81, Nos. 4/5, 188–93.

the gray corneal pannus infiltrate. Rapid healing of complicating corneal erosions and ulcers was the most spectacular improvement. The effect is not superior or more rapid than that obtained with sulfanilamide therapy, but penicillin may offer a solution in the treatment of sulfanilamide resistant or sensitive cases.

JOHNSTONE, I. L. **Penicillin Ointment for Out-Patients.** *Brit. Med. J.* 1946, Aug. 10, 201.

To dispense penicillin eye-ointments for use at home, a container is required which would not easily be contaminated by non-sensitive organisms and which would not affect the potency of penicillin. Collapsible metal tubes are unsuitable, as the metal would destroy the penicillin.

The author adapted glyco-gelatine capsules of about one gramme capacity, such as are used for other oculenta. After experimenting with different bases, he employed a mixture of vaseline, lanolin and liquid paraffin (30 per cent. of each), with distilled water making up the remainder. Penicillin was used in a strength of 500 Oxford units in the form of sodium penicillin (Glaxo).

Potency tests were carried out weekly at room temperature. No marked drop in inhibitory action on standard *S. aureus* occurred up to the seventh week, when the experiment was terminated by a "heat wave". Tests comparing the new B.P. base (which contains no water) with the above base are being undertaken. The base is free from undesirable irritation of the skin. The therapeutic effects in blepharitis are variable; but the author adds that more cases of blepharitis are cured clinically by thiazamide than by penicillin ointment at present. *H. J. O'D. Burke-Gaffney.*

YATOM, J. **An Outbreak of Conjunctivitis in Man associated with the Virus of Newcastle Disease.** *Harefuah.* Jerusalem. 1946, Feb. 1, v. 30, No. 3. [In Hebrew 57–8. English summary 58.]

"In October 1945 a small outbreak of conjunctivitis occurred in Mikveh Israel. Between 12/10 and 21/10 there were 17 cases. The cases, mostly women, were confined to individuals who had come in contact with chickens from a pen infected with Newcastle disease, *e.g.*, women and kitchen workers engaged in preparing the chickens for the table.

"The conjunctivitis was confined to one eye, usually the right, and there was no tendency to involve the second eye. There was no direct infection from one human being to another, no wife to husband infections. The outbreak stopped abruptly after the infected poultry had been destroyed.

"The incubation period in man was three days.

"The symptoms were:—inflammation and swelling of the conjunctiva of tarsi and sclera; at the commencement of the illness slight muco-purulent discharge; the cornea is not affected throughout the whole course of the disease, nearest lymphatic gland swollen and tender; acute symptoms last about three days; after 10 to 14 days complete disappearance of signs and symptoms.

"The clinical picture is reminiscent of conjunctivitis epidemica in Palestine but is distinguished by confinement to one eye and lack of corneal involvement."

WILSON, R. P. **A Statement on the Economic Importance of the Acute Ophthalmias of Egypt, with Recommendations for their Control.** *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 38–48.

EL-TOBGY, A. F. **A Scheme for Massive Abortive Treatment of Acute Ophthalmias by Chemotherapy.** *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 107–17.

NASHED, E. **Chemotherapy of the Acute Ophthalmias.** *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 129–32.

FAMILIARI, P. La cecità acquisita tra gli indigeni dell'A.O. problema clinico e sociale. [**Acquired Blindness in the Natives of East Africa : a Clinical and Social Problem.**] *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1945, v. 5, Nos. 5/6, 85–99. English summary (7 lines).

BLAND, J. O. W. **Spontaneous Folliculosis of the Conjunctiva in Baboons** (*Papio hamadryas*). *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 87–8.

BLAND, J. O. W. **Spontaneous Folliculosis of the Conjunctiva in Grivet and Vervet Monkeys** (*Lasiopyga griseoviridis* **Syn.** *Cercopithecus aethiops* **and** *L. pygerythra* **Syn.** *C. pygerythra*) **and the Susceptibility of the Grivet to Trachoma Virus.** *14th Rep. Memorial Ophthalmic Laboratory, Giza, Cairo for 1939–1944.* 72–86. [16 refs.]

CORRESPONDENCE.

In the abstract of the paper by BERNASCONI on trachoma [this *Bulletin*, 1945, v. 42, 488] the statement is made that " the author . . . treated a small number of incipient cases of trachoma with a preparation containing vitamins A and D by the mouth, without local applications." The author points out that 24 cases were treated, of which 9 were incipient and 15 in the granulomatous stage (2 having complications). The results were good. He also remarks that cod liver oil has been used with good effect as the source of vitamins A and D.—*Ed.*

TROPICAL ULCER.

i. NATH, T. **Tropical Ulcer in the Hissar District of the Punjab.** *Indian Med. Gaz.* 1946, Apr.–May, v. 81, Nos. 4/5, 169.

ii. LUTHRA, P. N. **Ulcus Tropicum in Fazilka (Punjab).** *Ibid.* 169–71.

i. It is pointed out by the author that whilst tropical ulcer has been reported in the humid climate of Assam and South India, and other parts of the country, no cases had been reported so far from the Punjab.

The paper discusses 60 cases seen in the Hissar district of the Southern Punjab, where rainfall is very low and the soil is mostly sandy.

The clinical, aetiological and microscopic features described by the author follow the now well-recognized picture. Two points of interest are that most cases occurred under the age of 18 years, and that they were most frequent between September and January.

Three groups of 20 patients were subjected to different treatments, namely :—(1) scraping, followed by dressings of 5 per cent. copper sulphate ; (2) scraping and sulphonamide powder ; and (3) strapping with leukoplast for at least 10 days.

The first two groups took about 35 days to heal, whilst the third did remarkably well and healed in 15 days.

The author noted scars on the lower limb in several patients examined for medico-legal identification, and presumed therefrom that the ulcer had existed in the district for a number of years.

ii. This author saw 106 cases of tropical ulcer in the out-patient department in Fazilka, a small town in the Ferozepore district of the Punjab with a population of 26,000. The climate is very hot and dry in summer and cold and dry in winter. The average rainfall is about 10 inches, but in 1944 it reached about 16 inches.

The author describes the disease as assuming a mild epidemic form after the unusually heavy rains in 1944, and " a milder epidemic was also noticed after the summer rains in 1945." [The use of the word " epidemic " is perhaps something of an overstatement.]

The description of the cases closely resembles that given by the author quoted in the preceding abstract. Over 50 per cent. of the patients were under 16 years of age, and 95 per cent. were under 30. Only 10 were females.

Twenty-eight hospital patients were treated by complete rest, and daily cleansing of the ulcers with a solution containing 3 drachms of copper sulphate, 1 drachm of carbolic acid and 1 ounce of water. The ulcers were then dressed with boro-iodoform. With this treatment, the average stay in hospital was 14 days. When half of the patients were treated with dressings of magnesium sulphate and sulphonamide powder in equal parts, the average stay in hospital was 20 days. Penicillin dressings, used in one case only, did not accelerate recovery.

The author's experience that the disease is one affecting the poor and ill-nourished agrees with that of the previous author writing from Hissar, and indeed with that of most observers ; and he sounds a note of warning that it may well spread and assume severe epidemic proportions under the serious food conditions now prevailing in India, a danger which health authorities and others should take anticipatory steps to meet.

[Although these papers have little new to add to the large *dossier* of tropical ulcer, the close resemblance between these independent testimonies in different parts of a Province, where tropical ulcer was not believed to occur, is significant ; and there is sound sense and humanity in the timely reminder for the need to take advance account of this additional potential complication of the already severely taxed vitality of the people in the famine-afflicted regions of India.]

H. J. O'D. Burke-Gaffney.

GOLDEN, A. & PADILLA, E. **Tropical Ulcer in Guatemala. Pathologic, Bacteriologic, Mycologic and Clinical Aspects.** *Arch. Pathology.* 1946, June, v. 41, No. 6, 612–30, 3 figs.

This paper represents the results of observations made by the authors on 24 patients suffering from tropical ulcer. All the patients originated in the lowlands—from sea level to 1,000 feet—and were predominantly American Indian and Indian-Caucasian, though all types in the lowlands were included, Indian, Caucasian and Negro.

The patients comprised members of age-groups from 11 to 56 years of both sexes. Sex and occupation did not appear to affect incidence significantly.

Full histories were obtained, and physical and laboratory examinations were made ; and the investigation included the dietary history of the patients. No common nutritional deficiency was noted.

Routine clinical laboratory tests revealed nothing of new importance, and bacteriological examination, including attempts to isolate fungi, were inconstant and added nothing to the list of recognized flora.

The authors exclude various causative factors in their series one by one, but place most significance on the biopsy findings. After describing the general

and usually recognized histological features of tropical ulcers, they note that the distinguishing feature in all cases was to be found in the vascular changes. These consisted essentially of thickening and narrowing of the lumina of vessels, which were occasionally obliterated and sometimes recanalized. These signs were found, to some extent, alike in arteries, veins and capillaries.

Detailed descriptions of these histological changes are given; and the authors believe that they are primary vascular changes and not secondary to inflammation. Arguments are given in support of this thesis, the chief of which are that (1) even the most severe vascular lesions were found in all cases at an early stage; (2) the most pronounced vascular changes were situated away from the zones of greatest inflammatory exudate; (3) the condition is entirely different from the endarteritis or vasculitis commonly encountered as secondary to inflammation.

In the absence of any other specific factors, the authors consider that the vascular feature stands out clearly, for it was common to all cases. They discuss the possible causes of these vascular changes, but were unable to find any one specific reason for the interference with local blood supply. They consider that treatment of tropical ulcer should aim especially at restoring the adequacy of the blood supply to the part.

[Among the vast number of factors which have been indicted as causes of tropical ulcer, there have frequently been included that of inadequate blood supply to the affected part. Where the authors' further confirmatory study is especially valuable is in the carefully described and well-illustrated histopathological investigations.] *H. J. O'D. Burke-Gaffney.*

MISCELLANEOUS DISEASES.

i. Manson-Bahr, P. E. C. & Charters, A. D. **Epidemic Thrombophlebitis in the East Africa Command.** *Lancet.* 1946, Sept. 7, 333–5, 3 figs.

ii. Fisher, A. C. & Lendrum, A. C. **Epidemic Thrombophlebitis.** [Correspondence.] *Ibid.* Sept. 21, 438.

i. The authors describe a syndrome of "recurrent thrombophlebitis accompanied by pyrexia, often relapsing and sometimes associated with stiff neck," among East African soldiers. No previous record of this condition was known to the authors, but they quote Gelfand who, in his book "The Sick African" refers to the condition of acute thrombophlebitis of unknown aetiology described in N. Rhodesia by Fisher [this *Bulletin*, 1942, v. 39, 199].

Occasional cases had been seen in the East African Command during 1941–1943, but in 1944 the disease began to assume epidemic proportions; and cases were observed independently at three different centres; most, but not all, of the 627 patients with this syndrome, admitted to two General Hospitals, had previously been treated for venereal disease at special centres.

The authors describe the clinical features observed in 145 cases; three varieties of the syndrome were seen:—(1) a short term fever with stiff neck, often followed by relapses; (2) thrombophlebitis affecting one or more limbs, tending to relapse; (3) pyrexia, usually relapsing, without evident phlebitis. As stated above, most of these patients had received treatment for venereal disease, which involved venepuncture, but 23 did not give a history of injection. The interval between the last injection and the onset of symptoms varied from three days to seven months. The first variety, short-term fever with stiff neck, developed either as the first phase of the syndrome or as a sequel to previous attacks of phlebitis; it lasted 2–30 days, usually about 4; it usually relapsed,

with stiff neck, or thrombophlebitis, but occasionally without either. There was no sign of cervical thrombophlebitis. Laboratory examinations, including some attempts at bacteriological culture, were negative, except for relative lymphocytosis in a few cases. The second variety, thrombophlebitis affecting one or more limbs, was either acute or subacute in most cases, but a chronic form, a form with involvement of the portal vein, and a form associated with arteritis, are described. Again, laboratory investigations were negative, and biopsy of the affected vein in 6 cases showed simple thrombosis only, with no evidence of inflammation of the wall of the vein. The veins of the legs were most commonly affected, but in 20 of 105 cases the veins of the arm were thrombosed. In no case was embolism noted.

In the third variety the pyrexia was irregular and lasted for 3–21 days, without evident phlebitis; in many of these cases the diagnosis was suggested by a history of recent anti-syphilitic therapy.

The authors discuss the differential diagnosis of these conditions.

The cause of this disease is still unknown. That it may be due to a virus, possibly transmitted during intravenous medication for syphilis, is suggested by the relative lymphocytosis, the negative bacteriological findings and the fact that the curve of incidence followed quite closely the curve of incidence of hepatitis which was related to administration of arsphenamine. No antibodies to Rift Valley fever were found in two patients examined. It is possible that the administration of arsenic or bismuth predisposed to the present syndrome, perhaps by rendering the veins susceptible to infection. There was no evidence that this thrombophlebitis was related to marasmus—most of the patients were well nourished. The authors note that thrombophlebitis is not common in the civilian Africans of the area.

ii. In this letter Fisher and Lendrum refer to the pathological findings in the veins in the Northern Rhodesia cases mentioned above.

"It has come to be appreciated (A.C.F.) that only a short stretch of the vein was primarily involved, and microscopy of this zone has now revealed a peculiar form of inflammation characterised by extreme proliferation of young capillaries in the disrupted media of the vein, and by the presence of phloxinophil intracytoplasmic inclusions." *Charles Wilcocks.*

JACOBY, H. **Curative Treatment of Lathyrism, a Disease of the Nervous System.** *Indian Med. Gaz.* 1946, v. 81, Nos. 6/7, 246–7.

The author reports favourably on prostigmine.

COX, C. D. & ARBOGAST, J. L. **Melioidosis.** *Amer. J. Clin. Path.* 1945, Dec., v. 15, No. 12, 567–70. [14 refs.]

This paper reports a case of melioidosis in a 27-year-old American infantry man, who died on the 8th day of disease. He had been 21 months overseas, 20 of which had been spent in Burma; the month preceding admission to hospital he had been located at Dinjan in Assam. Illness started acutely. There was a swinging temperature up to 105°F., liver and spleen were both palpable and scattered pustules appeared over the entire body. Clinical diagnosis was one of generalized septicaemia of undetermined aetiology. Post-mortem abscesses averaging 0·8 cm. in diameter were present in the skin, liver, spleen, lungs, lymph nodes, epididymis and testicle. They were most numerous in the lungs where they were becoming confluent. Histologically they were characterized by a central zone of coagulation necrosis surrounded by a haemorrhagic zone; numerous polymorphonuclear leucocytes were

present. A motile Gram-negative bacillus, isolated from the blood and skin lesions during life and from the lesions post-mortem, was identified as *Malleomyces pseudomallei* (*Pf. whitmori*). *S. P. Bedson.*

ASHLEY, P. **External Otitis in the Tropics.** *U.S. Nav. Med. Bull.* 1946, Aug., v. 46, No. 8, 1230–36, 4 figs.

Ashley states that the cases of external otitis which occur in ships operating in tropical waters may be divided into three categories.

In the first group the men complain of pain in the ear, discharge, partial deafness, itching or burning. The auditory canal is partly or wholly blocked with a cheesy or flaky mass consisting of cellular débris and moist exudate, in which purulent matter may occasionally be incorporated. If the meatus is cleaned, its walls are found to be raw, red and, sometimes, bleeding ; they may also be slightly oedematous.

Ashley recommends that the ear should be thoroughly cleansed, and if X-ray therapy is not available (he regards X-rays as the ideal treatment for all forms of external otitis) the meatus should then be swabbed with a 4 per cent. solution of boric acid in 50 per cent. ethyl alcohol. Thereafter a cotton wick impregnated with 10 per cent. aqueous solution of sodium sulphadiazine should be inserted and left in place. This dressing should be changed thrice daily. On the third day, if the walls are dry, the treatment is changed ; the toilet of the ear is carried out twice daily and a search is made to determine which of ten preparations—the formulae for which are given—will suit the patient best and complete the cure. The preparations vary from sulphanilamide powder to a lotion containing 1 gramme of benzoic acid, and 1 gramme of salicylic acid in 30 cc. ethyl alcohol. The ten preparations are shown below :—

	Gm. or cc.
Formula No. 1 :	
Salicylic acid	1·0
Ammoniated mercury	1·0
Rose water ointment	30·0
Mix.	
Formula No. 2 :	
Ammoniated mercury	1·0
Petrolatum	30·0
Mix.	
Formula No. 3 :	
Salicylic acid	1·0
Ethyl alcohol 95-per cent. to make solution	30·0
Formula No. 4 :	
Sulfanilamide powder.	
This is best instilled with a powder blower.	
Formula No. 5 :	
Sodium sulfadiazine ointment 5-per cent.	
This is a new item in the Supply Catalogue.	
Formula No. 6 :	
Sulfathiazole powder	1·5
Petrolatum q.s.ft.	30·0
Mix.	
Formula No. 7 :	
Salicylic acid	3·0
Ethyl alcohol 95-per cent. to make solution	30·0
Formula No. 8 :	
Thymol	0·6
Cresatin q.s.ft.	30·0
Mix.	

Formula No. 9 :

Ichthammol	1·2
Naphthalan	6·0
Zinc oxide	15·0
Starch	15·0
Petrolatum q.s.ft.	60·0

Mix.

(This preparation is not available at sea but is very effective in Group 3 cases.)

Formula No. 10 :

Benzoic acid	1·0
Salicylic acid	1·0
Ethyl alcohol 95-per cent. to make solution	30·0

" Benzoic acid was not available, but we feel that this so-called ' liquid Whitfields ' would be of use in some cases."

The second variety of otitis externa is more acute. The patient's temperature may be raised to 103°F. and the ear is exquisitely tender. The walls of the auditory canal may be oozing or dry, but they are very oedematous. This variety of otitis externa responds to rest in bed, oral administrations of sulphadiazine and the application of heat to the ear from a partly filled hot water bottle. After 24 hours the patient is usually so far recovered that he will submit to local therapy ; the meatus is then cleansed, and a wick impregnated with metacresyl acetate (cresatin) is inserted and renewed thrice daily. After some 3 or 4 days he may leave his bed, and local therapy continues as for cases of Group I.

The third group consists of patients who complain of itching and occasional slight pain in the ear. The walls of the canal are found to be dry and scaling. The condition may arise *de novo*, or may be a sequel to the other types of inflammation described above. Treatment consists in carrying out the toilet of the ear regularly, and using one or other of the ten applications which the author has listed for use in cases of Group I.

Ashley believes that the adverse climatic conditions of the tropics associated with the entry of water into the ear whilst the patient is bathing under a shower, or swimming, and the removal of wax from the ear with toothpicks and other implements, should all be considered as aetiological factors. In cases of Group 3 " the production of ' dry ' wax, or no wax or oil at all, in the ear canal leads to scaling and fissuring of the skin, giving the picture commonly known as ' eczema ' of the external ear canal. Fully developed this ' eczema ' is identical with the picture seen in Group 3 and may quickly develop into either of the other two types." *R. M. B. MacKenna.*

KEAN, B. H. & TUCKER, H. A. **Etiologic Concepts and Pathologic Aspects of Ainhum.** *Arch. Pathology.* 1946, June, v. 41, No. 6, 639–44, 1 fig. [33 refs.] [Refs. in footnotes.]

The authors group the aetiological factors comprising the many theories as to the causation of ainhum under three general headings : infectious, constitutional, and mechanical.

In this paper, they discuss the literature under these general heads and then describe the records of 45 sufferers from ainhum on the Isthmus of Panama, 12 of whom they had observed clinically.

All the patients were negro males and the little toe was the only digit involved. A deep encircling groove separated the toe from the remainder of the tarsus at the level of the digito-plantar fold. In advanced cases, the " constriction " progressed until only a thin pedicle kept the small " potato-like " toe to the foot.

The histopathological features conformed to the recognized descriptions of ainhum ; but the authors consider that in published reports, undue significance is commonly placed on a number of features ; and they express the following opinions :—

(1) The groove is not caused by a *constricting band* of fibrous tissue.

(2) Identical epithelial changes may be seen in the toes of those not suffering from ainhum.

(3) The vascular changes noted are not specific to ainhum.

(4) The nerve and gland changes do not appear significant. The authors found it difficult to evaluate the atrophy of bone and the subcutaneous inflammatory reactions, which seemed to be significant ; but the evidence that the essential lesion of ainhum might be chronic osteomyelitis was not impressive, although the low-grade cellulitis and fibrosis of the corium were more suggestive.

The authors conclude that the cause and pathogenesis of ainhum are not clear, but that the racial factor seems most important.

H. J. O'D. Burke-Gaffney.

HERSH, J. **Ainhum : Report of a Case.** *J. Internat. College of Surgeons.* 1946, July–Aug., v. 9, No. 4, 477–81, 2 figs.

SANYAL, S. C. **Pedunculine (for the Treatment of Bengal Splenomegaly).** *Indian Med. Gaz.* 1946, June–July, v. 81, Nos. 6/7, 242–3.

CAWSTON, F. G. **Some Risks from New Remedies in Tropical Disease.** Reprinted from *Med. Press & Circular.* 1946, May 29, 361–2.

GENERAL PROTOZOOLOGY.

PLAUT, A. **The Problem of Human Toxoplasma Carriers.** *Amer. J. Path.* 1946, Mar., v. 22, No. 2, 427–31, 6 figs. on 1 pl.

In the case of a boy who died of severe jaundice, associated with enlargement of the liver and spleen and ascites, and also in the case of a woman who died of peritonitis following operation for intussusception, bodies presumably parasitic in nature were found in the myocardium. In the first case two masses were seen in enlarged muscle-fibres. Each mass, which had a fairly distinct outline, consisted of numerous closely-packed particles about 1μ in diameter. In the second case, a single group of somewhat larger radially arranged bodies, each with a central nucleus, was seen. In neither case was there evidence of parasitic invasion elsewhere in the body. Slides and photographs were submitted to a number of experts, who failed to agree as to the significance of the structures, suggesting, among other things, that they were sarcosporidia, toxoplasma or contaminants. The author calls attention to the organisms, which are illustrated in photographs, because of their importance from the point of view of the discovery of carriers of toxoplasma infection. *C. M. Wenyon.*

GENERAL ENTOMOLOGY.

BEADLE, L. C. **An Ecological Survey of some Inland Saline Waters of Algeria.** *J. Linnean Soc.-Zoology.* 1943, v. 41, No. 278, 218–42, 1 fig. [38 refs.]

This paper is the result of an expedition to study the saline inland waters in Algeria in the winter of 1938. They ranged in density from 1,000 to 1,172

(fresh water=1,000, sea water 1,022). Analyses for the various ions were made, *p*H values, alkalinity and other data are given. The fauna and flora found can be divided into three groups :—(1) freshwater species only found in low salinities ; (ii) forms only found in intermediate salinities ; (iii) forms capable of living in extremely saline waters. Many interesting and useful results of ecological importance are given, but few forms of direct medical importance were found. *Aëdes detritus* and *Aëdes caspius*, which are confined to saline waters, were recovered several times. *Anopheles multicolor* was found once in stagnant water having a density of 1,008. *Kenneth Mellanby.*

WOLFS, J. Note sur les moustiques de Coquilhatville. (Deuxième note.) [**A Note on the Mosquitoes of Coquilhatville. Second Note.**] *Ann. Soc. Belge de Méd. Trop.* 1946, June 30, v. 26, Nos. 1/2, 95–104, 1 fig.

HILL, R. B. & HILL, Claire M. **A List of the Mosquitoes found in Jamaica.** *Catalogus Insectorum Jamaicensis. Supplement.* 1945, 3 pp. Dept. of Agriculture, Jamaica. Kingston : Govt. Printer.

The compilers of this list have made extensive collections of mosquitoes in various parts of Jamaica, and in the past three years have found one new species and 13 others hitherto unreported from the Island. Three other species, new to Jamaica and found by other workers, are included. A total of 50 species is listed, and the list includes many mosquitoes of medical importance.

H. J. O'D. Burke Gaffney.

WANSON, M. & LEBIED, B. Un nouvel Anophèle cavernicole du Congo belge. *Anopheles* (*Myzomyia*) *vanhoofi* spec. nov. [**A New Cavernicolous** *Anopheles* **from the Belgian Congo.**] Reprinted from *Rev. Zool. Bot. Afr.* 1945, Dec. 29, v. 39, No. 1, 118–29, 19 figs.

In the lower part of the Belgian Congo near Thysville there is a very large cave which is known to extend at least a mile from its entrance. It contains a remarkable fauna, including a blind fish, cave crickets and so forth, also two giant species of *Phlebotomus*.

The authors here describe a new species of *Anopheles* from this cave. The early stages have been found in total darkness in a chamber some three hundred yards from the entrance. They occur in small basins in the rock which are filled with water that drips from the roof ; the adults are found in the same place, and it is thought that they never leave the cave, but feed on certain bats (*Miniopterus*) which are abundant.

The adult mosquito is large and dark without ornamentation on the legs or palps and with only some very obscure pale spots on the wings : there are small differences in the wing markings between the male and female. The morphological characters of the larva and adult indicate clearly that it is a *Myzomyia* and that it probably belongs to the group *Eomyzomyia* with affinities to *A. wilsoni*. [So far as we are aware this is the first record of a completely cavernicolous mosquito.] *P. A. Buxton.*

KNIPE, F. W. **Improvements in Equipment for killing both Larvae and Adult Mosquitoes with DDT.** Reprinted from *Agric. Engineering.* St. Joseph, Michigan. 1945, Nov., v. 26, No. 11, 459–62, 464, 468, 5 figs.

The introduction of DDT involves changes in spraying equipment because the dosages applied and the degree of atomization required differ from those of preparations formerly used.

A hand gun of the " Cobra " type has been developed which will operate at pressures between ½ and 1 atmosphere (7–14 lb./sq. in.). The orifice is 1 mm. in diameter, and with a working pressure of 0·5 kgm./cm² (½ atmosphere) this will deliver 2·5 cc. of DDT-oil solution per minute. This permits easy coverage of an acre with one quart of oil. The same type of nozzle can be used without the hand plunger pump by attachment to an independent air supply. The nozzle can be adjusted so that, at 1 atmosphere pressure, it will deliver from 2·5 to 110 cc. per minute. Up to 54 cc. an excellent " pyrethrum type " spray is produced [? atomized mist] and just above this, a wet spray is formed which is suitable for treating walls.

A small hand operated air-pump has been devised for use with the sprayers. This is operated by a lever in the same way as the ordinary knapsack oil-spray pump. It develops a pressure up to 2½ atmospheres. It can either be used in conjunction with a knapsack tank or the air can be delivered to a hand sprayer with a container. Two such pumps strapped on a brace on the back of an operator will supply air for a single spray-gun carried by another operator. This arrangement is convenient for indoor spraying where the sprayer has to be manoeuvred over furniture, etc.

Types of spray patterns are discussed. The fan-shaped spray is considered preferable to the round spray for careful treatments in furnished houses. Fan-shaped sprays can be produced by suitable nozzles, both with air-atomized and pressure-atomized (whirling jet) sprays.

A convenient extension arm can be made from 6 mm. aluminium or copper tubing led through bamboo poles drilled lengthwise. An extension of this type four to five metres long is not too heavy and is useful for spraying high barns, etc. *J. R. Busvine.*

DAVID, W. A. L. **The Quantity and Distribution of Spray collected by Insects flying through Insecticidal Mists.** *Ann. Applied Biol.* 1946, May, v. 33, No. 2, 133–41, 2 figs. & 1 pl. [12 refs.]

If a housefly or a mosquito (*Aëdes aegypti*) flies through a mist of finely divided insecticide, droplets accumulated in transit can be seen by subsequently examining the wings. On other parts of the body, oil-carried insecticides spread rapidly, but by use of an aqueous spray, droplets can also be found on the antennae, eyes, mouthparts, large body bristles, the halteres and on fine hairs guarding the anterior spiracles. If a spray mist is produced with oil dyed with Sudan III, and the flies are dissected at intervals after exposure, it is found that the dye penetrates the integument, is extracted from the haemocoel by the Malpighian tubules, and excreted through the anus. Houseflies with mouth and anus blocked were found to show large quantities of dye in Malpighian tubules and rectum one hour after spraying.

A colorimetric method is described by which the quantity of spray drops picked up during flight can be estimated. With both *Musca* and *Aëdes*, about three times as much spray is deposited on the wings as on the body. With insecticides which do not cause paralysis, much of this deposit is removed during the course of twenty-four hours. Deposit on the wings is removed by cleaning movements of the legs and there is also a general absorption and excretion. Penetration of the cuticle does not appear to occur in dead insects.

The quantity of spray accumulated depends on the physical qualities of the mist, especially on the size and number of drops. Much more is accumulated directly after spraying than in an equivalent period with a mist four to ten minutes old. The addition of a small quantity (5 per cent.) of a non-volatile oil reduces evaporation of the droplets, which therefore remain of large size ; as a consequence, many more impact on flying insects.

By the quantitative method it is possible to estimate the median lethal dose of different insecticides as deposited from spray mists. Thus :—

Insecticide	Median lethal dose (mgm. per kgm. live weight)			
	Musca (3–5 days old)		*Aëdes* (1–4 days old)	
	Males	Females	Males	Females
DDT (0·3 per cent. sol.)	6·0	9·0	5·5	8·0
Gammexane (0·3 per cent. sol.)	2·0	3·0	3·0	3·5
Pyrethrins (0·1 per cent. sol.)	—	—	0·5	1·0
Pyrethrins (2·0 per cent. sol.)	31·0	38·0	—	—

J. R. Busvine.

POTTER, C. & GILLHAM, E. M. **Effects of Atmospheric Environment, before and after Treatment, on the Toxicity to Insects of Contact Poisons. I.** *Ann. Applied Biol.* 1946, May, v. 33, No. 2, 142–59, 7 figs. [16 refs.]

The authors discuss the influence of temperature and humidity on precise laboratory testing of insecticides. Experiments are recorded in which the effects of keeping an insect cool or warm, before and after spraying, are related to the mortality caused by different contact insecticides in various media. The insect used throughout was the flour beetle *Tribolium castaneum*, which was reared at 80°F. (27°C.).

It was found that exposure of the insects to cool conditions (50–60°F.) or warm conditions (80°F.) for one to twenty-four hours before spraying did not have a great effect on the results. However, insects kept under warm conditions after spraying were much more resistant to most of the insecticides tested than those kept under cool conditions. The actual ratios of potency of the different insecticides were as follows :—

Poison	Medium	Ratio (potency of insecticide with cold storage after treatment as proportion of that under hot)
Pyrethrins	Aqueous	2·67 and 5·01 (2 expts.)
Pyrethrins plus Terpineol	,,	7·21 and 6·90
Lauryl thiocyanate	,,	1·43
Nicotine	,,	1·23
Dinitro-*o*-cresol	Ethylene glycol	1·46
DDT	Oil	2·61
Wakefield white oil	—	0·87

It will be observed that the only spray which caused greater mortality under warm conditions was the chemically inert Wakefield white oil.

It is believed that this effect of temperature must be referable to the physiological condition of the insect rather than to any direct physical action on the insecticide. *J. R. Busvine.*

LARSEN, Ellinor B. & THOMSEN, M. **The Influence of Temperature on the Development of some Species of Diptera.** Reprinted from *Vidensk. Medd. fra Dansk naturh. Foren.* 1940, v. 104, 75 pp., 18 figs. [39 refs.]

It is difficult to obtain accurate figures for the rate of development of flies which breed in dung, and the optimum conditions cannot be precisely defined. Even when experiments are carried out in a thermostat, fermentation may raise the temperature of the medium, or evaporation may lower it, and so invalidate the results. This paper is an account of very extensive experiments in which every attempt was made to eliminate these sources of error. Five species of diptera whose larvae live in dung were used: *Musca domestica, Stomoxys calcitrans, Lyperosia irritans, Haematobia stimulans, Scatophaga stercoraria.*

With *Musca domestica*, the minimum duration of the pre-imaginal period was 6·92 days at 33°C. Above this temperature there was some retardation, and 40°C. was too hot, causing 100 per cent. mortality. Development was slowed down by temperatures below 33°C., to 15·66 days at 21°C. and 54 days at 14·2°C. The "developmental zero" was in the region of 12°C., though some development occurred at as low a temperature as 9·2°C. The various stages of development—egg, feeding larva, pre-pupa, pupa—were affected in a roughly proportionate manner.

Stomoxys calcitrans developed most rapidly at 31·4°C., with a pre-imaginal period of 10·75 days. The rate for the whole zone of temperature from 29°C. to 33°C. closely approximated to this figure. Above 33°C. there was a marked retardation, and 35°C. proved fatal. At 15·7°C. development took 44 days. The developmental zero was about 12°C. There was no indication of a diapause occurring in either of the above species.

Lyperosia irritans was more difficult to rear in the laboratory. The shortest duration of the pre-imaginal period was 7·54 days, at 32·3°C. A sudden drop in temperature, below 18°C. seemed often to induce a pupal diapause.

Haematobia stimulans is also difficult to rear in the laboratory. This species was adversely affected by much lower temperature than the others, a fact probably correlated with its scarcity during the summer and its commonness in spring and autumn. The maximum rate of development (pre-imaginal period 11·1 days) occurred at 28·3°C. The threshold of development was at about 9°C. A diapause may possibly occur during development.

Scatophaga stercoraria was found to be even more easily harmed by high temperatures. The shortest pre-imaginal development, 15·58 days, was found at 25·3°C. At 27°C. and above, all were killed. The threshold of development was about 2°C. *Kenneth Mellanby.*

SWEETMAN, H. L. **DDT as a Spot Treatment for Flies.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 380–81. [13 refs.]

The author observes that if one is treating buildings with DDT in order to reduce domestic flies, it is sufficient to spray the particular places where flies settle or concentrate. He says that he has used "25% DDT in white or odourless kerosene" [which is not possible: it is about five times more than a saturated solution]. *P. A. Buxton.*

BRUCE, W. G. & BLAKESLEE, E. B. **DDT to control Insect Pests affecting Livestock.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 367–74, 1 fig.

UNSWORTH, K. & GORDON, R. M. **The Maintenance of a Colony of** *Phlebotomus papatasii* **in Great Britain.** *Ann. Trop. Med. & Parasit.* 1946, July, v. 40, No. 2, 219–27, 3 figs. [24 refs.]

Several workers have maintained cultures of *Phlebotomus papatasi* in tropical and subtropical countries, but there has been no strain maintained for long in this country notwithstanding the medical importance of the species. The "classical" methods essentially involved keeping the immature stages at the correct humidity, and proper feeding of the adults on blood. By means of such technique, five generations of *P. papatasi* have now been bred in Britain, with a continually increasing population. However, this method was so time-consuming that a more automatic technique has been devised. The breeding chamber in this case is inside a large block of porous cement, which is soaked in water and thus ensures the correct humidity. Each day a baby rat, strapped to a board, is inserted to feed the adults. The larvae live in soil and in rabbit faeces at the bottom of the breeding chamber. This technique has been successful, the culture being maintained with the minimum of supervision, though it is as yet not possible always to ensure the production of very large numbers of flies. *Kenneth Mellanby.*

KIRK, R. & LEWIS, D. J. **Taxonomy of the Ethiopian Sandflies** (*Phlebotomus*). **II. Keys for the Identification of the Ethiopian Species.** *Ann. Trop. Med. & Parasit.* 1946, July, v. 40, No. 2, 117–29, 56 figs.

The paper is without doubt valuable as a summary of the present state of knowledge. In all there are rather over forty known species from Africa south of the Sahara. The authors have excluded a number of other names on the ground of wrong identification or because they are synonyms.

The body of the paper consists of keys of adult males and females of the three subgenera, *Phlebotomus*, *Sintonius* and *Prophlebotomus* : it is assumed that one knows the subgeneric characters and can refer a specimen to one or another of these. *P. A. Buxton.*

DAMASCENO, R. G., CAUSEY, O. R. & AROUCK, Regina. Estudos sobre Flebotomus no Vale Amazônico. Parte V. Descrição de *F. williamsi*, *F. deanei*, *F. carvalhoi*, *F. lopesi*, *F. castanheirai*, *F. fariasi*, *F. baityi* e *F. campbelli*. (Diptera-Psychodidae). [**Studies on** *Phlebotomus* **in the Amazon Valley. Part V.**] *Mem. Inst. Oswaldo Cruz.* 1945, Aug., v. 43, No. 1, 1–30, 47 figs. (39 on 8 pls.) [14 refs.]

D'ANDRETTA, M. A. V. & D'ANDRETTA, C., Jr. As espécies neotropicais da família *Simuliidae* Schiner, 1864 (Diptera-Nematocera). I.—*Simulium* (*Eusimulium*) *orbitale* Lutz, 1910, *Simulium* (*Eusimulium*) *pintoi* n. sp. e *Simulium nigrimanum* Macquart, 1837, *sp. inquirendae*. [**South American Species of the Simuliidae.**] *Mem. Inst. Oswaldo Cruz.* 1945, Aug., v. 43, No. 1, 85–152. Numerous figs. on 19 pls. & 2 maps. [18 refs.] English summary (7 lines).

DENNELL, R. **A Study of an Insect Cuticle: the Larval Cuticle of** *Sarcophaga falculata* **Pand. (Diptera).** *Proc. Roy. Soc.* Ser. B. 1946, Aug. 7, v. 133, No. 872, 348–73, 7 figs. (3 on 1 pl.) [58 refs.]

MALDONADO CAPRILES, J. **The Fleas of Puerto Rico.** *Puerto Rico J. Pub. Health & Trop. Med.* 1945, Dec., v. 21, No. 2, 173–83, 8 figs. [Refs. in footnotes.] [Spanish version 184–92.]

SWEETMAN, H. L. **DDT to control Cat and Dog Fleas and Dog Lice.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 417–18.

BUSHLAND, R. C. **Insecticides applied to Forest Litter to control New Guinea Chiggers.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 344–7.

Since DDT, when sprayed from the air or otherwise, was successfully used to control mosquitoes over a wide area, many attempts have been made to control other vectors of diseases in a similar manner. The experiments carried out by the author in New Guinea were made against two species of trombiculid mite, *Schöngastia pusilla* Womersley and *S. blestowei* Gunther; they cause "scrub itch" but have not been proved to carry the rickettsia of scrub typhus. It is believed, however, that scrub typhus vectors would respond in a similar way.

An area in the New Guinea forest where mites abounded, so that up to about thirty could be collected from the boots in five minutes when a man crouched on the ground, was treated partly with 40 U.S. gallons [40 U.S. gallons=32 Imperial gallons] and partly with 80 U.S. gallons of 5 per cent. DDT in diesel oil per acre, and also partly with similar quantities of diesel oil alone. Other tests were made with 5 per cent. diphenylamine and 5 per cent. diphenylene, both at the rate of 80 gallons per acre. The ground was also dusted with 5 per cent. DDT in calcium carbonate, 320 pounds being applied to an acre.

The results of all these tests were similar. There was a temporary cessation of activity, but living mites were always found within three days of spraying, and within about a week normal activity had been restored. The diesel oil alone was quite as successful as any of the insecticides. [It should be noted that the rate of application of DDT was several hundred times as great as has been shown to be effective against mosquitoes, and yet it had no noticeable effect on the mite population.] *Kenneth Mellanby.*

SNYDER, F. M. & MORTON, F. A. **Materials as Effective as Benzyl Benzoate for impregnating Clothing against Chiggers.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 385–7.

Dimethyl phthalate, dibutyl phthalate and benzyl benzoate, when used to impregnate garments, have all been shown to be effective in preventing trombiculid mites from attaching to man. Dibutyl phthalate and benzyl benzoate have the advantage that they remain effective after the clothes have been washed [see this *Bulletin*, 1946, v. 43, 922, 923]. Extensive tests have been made at Orlando in Florida, to find other substances equally or more effective, and resistant to washing. Hundreds of substances have been tried, and discarded when either ineffective, easily washed out or irritating to the skin. Uniforms were impregnated with the chemicals at the rate of 2 grammes per square foot (about 8 per cent. of the weight of the cloth) and the wearers then sat or lay on the ground in an area abounding in mites (*Eutrombicula spp.*). Volunteers wearing untreated uniforms showed several hundred mite attachments.

The following substances were at least as effective as benzyl benzoate:—benzil; phenyl benzoate; benzoic acid 3, 5-dimethyl phenyl ester; 1, 2, 5, 6-tetrahydro-*o*-methyl benzoic acid, 1, 2, 5, 6-tetrahydro-*o*-methylbenzyl ester; phenaxathin; *p*-methoxydiphenyl; benzoic acid, alpha methylbenzyl ester; benzoic acid, phenyl-ethyl ester; benzoic acid, 2-chlorophenyl ester.

Kenneth Mellanby.

WOMERSLEY, H. **A Revision of the Microtrombidiinae (Acarina, Trombidiidae) of Australia and New Guinea.** Reprinted from *Records of the South Australian Museum.* 1945, June 30, v. 8, No. 2, 293–355, 38 figs.

Previous work by WOMERSLEY and HEASLIP [see this *Bulletin*, 1945, v. 42, 324] has done much to elucidate the relationships of the Trombiculid mites

which cause scrub itch and which carry scrub typhus in the Austro-Malayan and Oriental regions. The present paper deals with the sub-family Microtrombidiinae. These mites are not themselves of medical importance, but they are often confused with mites which are, and their description will prevent much unnecessary field work by medical investigators. Eleven genera and forty species are described. *Kenneth Mellanby.*

BERTRAM, D. S. **An Apparatus for collecting Blood-Sucking Mites.** *Ann. Trop. Med. & Parasit.* 1946, July, v. 40, No. 2, 209–14, 2 figs.

The mite *Lyponyssus bacoti* has been successfully bred in the laboratory [see this *Bulletin*, 1946, v. 43, 1080 and below]. One difficulty has been to isolate the mites from the sawdust and earth used as bedding for the cotton rats, which are used to feed the mites. The apparatus here described consists of a sample tube to hold the infested sawdust, etc. An electrically-heated coil moves slowly along the tube, and this drives the mites before it into a collecting tube. The whole is mounted on a trolley and works automatically. The heated coil moves at a rate of rather less than half an inch a minute, and so takes from one to two hours to traverse the three-foot sample tube. The operator need only attend to the apparatus at the start and finish of the operation. Substantial numbers of mites may be collected; in a typical case 113 mites, of which 60 were females, were isolated. Practically all adults are evidently collected, some nymphs are found, but larvae (which are relatively inactive) are not. The eggs are presumably destroyed by the heat. *Kenneth Mellanby.*

BERTRAM, D. S., UNSWORTH, K. & GORDON, R. M. **The Biology and Maintenance of *Liponyssus bacoti* Hirst, 1913, and an Investigation into its Role as a Vector of *Litomosoides carinii* to Cotton Rats and White Rats, together with some Observations on the Infection in the White Rats.** *Ann. Trop. Med. & Parasit.* 1946, July, v. 40, No. 2, 228–54, 12 figs. on 1 pl. [20 refs.]

The blood-sucking mite *Liponyssus bacoti* has been shown to be a vector of filariae, and also to be easily reared in the laboratory [see above]. This paper describes a similar technique for bearing the mites, with full descriptions of the means used for handling individual specimens for transmission experiments.

The life-cycle of the mite is described in detail, and illustrated with photomicrographs. The life-cycle consists of the following stages: adults of both sexes; the egg, which develops parthenogenetically in the absence of males; the six-legged larva, which moults without feeding; the "protonymph", a blood-sucking stage; the non-feeding "deutonymph". The whole cycle can be completed in ten days, and the mites breed so rapidly that the infestation may kill the host.

Adult mites fed on rats infected with the microfilariae of *Litomosoides carinii* often become themselves infected, though only a small proportion of the worms develop to later stages. Nymphs appear not to become infected.

The cotton rat has previously been shown to be easily infected by *Litomosoides* transmitted by *Liponyssus*. The white rat may also be infected, and microfilariae have been found in the pleural cavity and the heart blood, though not in the peripheral circulation, seventy-four days after exposure. However, many of the worms in white rats, as opposed to cotton rats which are easily infected, were dead, fragmented and encapsulated, and a marked cellular reaction was observed in the host. It appears probable that the white rat may therefore be unsuitable for chemotherapeutic research.

Kenneth Mellanby.

COOLEY, R. A. **Note on the Tick,** *Ixodes angustus* **Neumann.** [Research Notes.] *J. Parasitology.* 1946, Apr., v. 32, No. 2, 210.

This tick has been found to bite man ; it has not been incriminated as a transmitter of human disease.

SMITH, C. N. & GOUCK, H. K. **Observations on Tick Repellents.** *J. Econom. Entom.* 1946, June, v. 39, No. 3, 374–8.

The authors describe experiments in which men were exposed under natural conditions in places in the Southern United States in which ticks (generally *Amblyomma*) were abundant. The clothes were treated with a considerable number of repellent and insecticidal substances. It was found that dimethyl phthalate, benzyl benzoate, and Indalone were generally fairly effective for a period of several weeks. Pyrethrum in kerosene was effective, but for a much shorter period ; DDT appeared to be without effect. Untreated controls were observed in each experiment : the dose of repellent is not stated.

The work was then extended to considerable field tests, the numbers of men in each not being stated. These experiments seem to show that against the same tick, Indalone and dimethyl phthalate are the most effective of the substances tested. A high degree of protection is obtained if one treats only the edges or apertures in garments, but complete protection was seldom or never obtained. The repellents are more lasting if put on garments than on skin.

P. A. Buxton.

LABORATORY PROCEDURES.

BENDA, R. & FRANCHEL, F. Injections intra-medullaires osseuses. [**Injections into the Bone Marrow.**] *Semaine des Hôpitaux de Paris.* 1946, Sept. 7, v. 22, No. 33, 1571–7, 8 figs. [45 refs.]

The authors list a number of substances for injection which may be given by introduction through the bone-marrow. They describe the methods employed and state that this route is " equivalent to the intravenous route ".

REPORTS, SURVEYS, AND MISCELLANEOUS PAPERS.

KAUNTZE, W. H. **Social Medicine in the Tropics.** *Practitioner.* 1946, Aug. v. 157, No. 928, 123–30.

The points made by the Chief Medical Adviser to the Secretary of State for the Colonies, in this paper, are as follows. The standards of living, diet and housing of the indigenous people of most of the Colonies are poor, especially in the urban areas. Even in rural, agricultural areas the crops grown, which may be suitable to local climatic conditions, are quite often not nearly so suitable to human needs, and malnutrition is frequently present in the people. Housing is usually bad, water supplies are contaminated and sanitation primitive. The people, therefore, though prolific are unhealthy and consequently apathetic, and there is high mortality.

The diseases from which these people suffer are either those spread throughout the community—for instance, insect-borne, water-borne or infectious diseases —and those which are not necessarily transmissible from one person to another. In the early days of the medical services in the Colonies, emphasis was naturally laid on curative medicine, and of this there was more than enough to occupy the whole time of the medical staffs; but as the transmission of the great tropical diseases became more clearly understood, the emphasis gradually shifted towards prevention. It is the author's argument that the attitude of the medical services should more and more lean in this direction.

Prevention has been attempted in different ways—by campaigns against individual diseases (which have often failed because they were not integrated or continued), or by intensive campaigns against all diseases. For the latter a team of workers concentrates on a limited area, introduces protected water supplies, conservancy systems, control of disease-carrying insects, good housing, efficient agriculture, and gives such treatment as may be necessary. Success depends on the co-operation of the people themselves, and before this can be obtained they must be educated to understand, and believe in, the efforts made. When the intensive work has been completed, the team moves elsewhere, and the permanent staff undertakes the maintenance of the new conditions. Success in these schemes depends upon the local doctor, the local nurses, and their assistants, who are most closely in touch with the people, and who can stimulate and maintain interest in the general welfare; but these doctors, nurses and assistants need training in social medicine and in the appreciation of the influence of total environment on health.

[Readers will be reminded of several tendencies in the development of measures to improve the health of tropical peoples, which have become apparent in recent decades. Health units have been developed (not least by the author) in Central and South Africa, Mauritius and Ceylon; the great undertaking of the Anchau Settlement in Nigeria has proved its worth; and the Foréami service of the Belgian Congo has for long given a stimulating lead. It is surely on the lines here indicated that progress must be sought.] *Charles Wilcocks.*

MANSON-BAHR, P. **Tropical Medicine in the British Empire. Suggestions for the Future.** *Nature.* 1946, June 29, 858–60.

In this paper, in which Sir Philip Manson-Bahr points out that as a result of the war the number of patients returning to Britain with tropical diseases is very high, two constructive suggestions are made. The first is that, as an aid to teaching, there should be created a central institution at which strains of tropical parasites and their intermediate hosts should be maintained so that an abundant supply of teaching material could be available for the various schools at which tropical medicine is taught. This institution would be a miniature zoological garden, with hot-houses, animal houses, stables and laboratories; the site could be in some rural spot within reach of London. Snails, *Cyclops*, mosquitoes and flies, and many other animals could be bred and maintained under optimal conditions.

The second suggestion is that since there is in Britain a paucity of pathological specimens of tropical interest, whereas the laboratories of the great tropical centres—Colombo, Rangoon, Singapore, Shanghai and Hong Kong—possess ample materials, an expedition should be sent to visit these and other places to arrange for proper collection and despatch of specimens to the British schools of tropical medicine.

[It would seem that these very practical suggestions should receive careful consideration; if they were adopted, their value to teachers might well be enormous.] *Charles Wilcocks.*

MANSON-BAHR, P. **The Post-Graduate Teaching of Clinical Tropical Medicine.** Reprint of Chapter XVII, pp. 361–73 from Albert Schweitzer Jubilee Book. 1946. London: Jas. Clarke & Co. Ltd., Wardrobe Place, Carter Lane, E.C.4.

In this short contribution to the Albert Schweitzer Jubilee Book the author sets out some of his views on the influence of the recent war on medical teaching and on the steps which could be taken to improve the teaching of tropical medicine in Britain. He refers especially to the needs of students from abroad. His remarks on the collection of material in tropical countries are similar to those already recorded. *Charles Wilcocks.*

BOYD, W. S., STUBBS, T. H. & WEINSTEIN, P. P. **The Tropical Disease Education Program of the United States Public Health Service.** *Pub. Health Rep.* Wash. 1946, May 17, v. 61, No. 20, 707–11.

The most important diseases brought to the United States by men returning from tropical war service are thought to be malaria, amoebiasis, filariasis, hookworm infection, leishmaniasis, schistosomiasis and hydatid disease. The American Society of Tropical Medicine has therefore requested the Surgeon General of the U.S. Public Health Service to consider :—

" (*a*) The organization of several teaching teams to visit State and other laboratories for the instruction of personnel in techniques of the laboratory diagnosis of the common tropical diseases.

" (*b*) The dissemination of appropriate articles on the diagnosis and treatment of tropical diseases through its district directors to the practicing physician by means of State and local public health and medical publications.

" (*c*) The formation of a library of teaching films on tropical diseases to be loaned to medical societies and other appropriate organizations."

As a result a Tropical Disease Education programme has been developed in Atlanta, and plans have been made to offer intensive training in the diagnosis of parasitic diseases to technicians already employed by State, and local health department laboratories. The course lasts six weeks, of which the first two are devoted entirely to the microscopical diagnosis of malaria, and the remainder to the study of other parasites as they are found in clinical human material. This course is to be repeated every three months so long as it is needed, and will be flexible to meet particular needs. An extension service has also been devised, by which stained specimens are sent from time to time to 105 laboratories in 43 States, so that the accuracy of the technicians may be checked. Specimens are also sent to teaching centres. The Diagnostic and Training Centre at Atlanta now also offers a consultative diagnostic service.

A second objective of this service is to further the instruction of medical men in the subject of tropical medicine, and for this purpose it has concentrated upon the development of visual materials—photographs, lantern slides and films—to be used in teaching centres and at meetings of medical societies.

Charles Wilcocks.

ANDREWS, J. M. **The United States Public Health Service Communicable Disease Centre.** *Pub. Health. Rep.* Wash. 1946, Aug. 16, v. 61, No. 33, 1203–10.

This seems to be an account of the organization and programme of the Centre in Atlanta referred to in the preceding abstract, though the names given to the centres are not the same. The Communicable Disease Centre has taken over the functions of the Office of Malaria Control in War Areas, and deals largely with diseases common in the tropics and transmitted by insects ; the author

points out that very large numbers of Americans, infected with these diseases, are returning or have returned from military service in the tropics, and that there is a constant danger of the introduction of infected persons, or of non-indigenous vectors, into parts of the United States in which the diseases could spread or the vectors multiply. Moreover, when the need for medical men and others skilled in parasitology and tropical diseases was felt during the recent war, the number available was small.

The development of the centre is summarized in three categories :—(1) Training and training-aid production ; (2) Epidemiologic and laboratory services ; (3) Operational services. Training in the control of malaria, typhus, plague etc., and in the diagnosis of these infections, is offered to State and local health officers ; it includes the usual methods and the production of articles, charts, exhibits, lantern slides and films. The epidemiological and laboratory services provide the means for instruction and for investigation of outbreaks of disease ; mobile laboratories are available. One function will be the evaluation of techniques. The operational services will deal with the control of diseases and the testing of material and equipment, including insecticides. These services will be available for emergency control, and will also provide demonstrations of methods in appropriate areas.

The author notes that by 1945 upwards of half a million American soldiers had contracted malaria, chiefly due to *P. vivax*, and that these men, diffused, on return, over the whole country, could well establish the disease in new communities ; important existing foci were therefore more intensively controlled. The other possible infections in returning soldiers are also considered in relation to spread within the United States.

Most of the expenditure (70–80 per cent.) of the Malaria Control in War Areas organization has been accounted for by labour. Special investigations of operational significance were carried out in association with the National Institute of Health, the Tennessee Valley Authority, and other organizations. These wartime operations are rapidly diminishing, but it is felt that a basic organization of physicians, engineers and biologists should be retained, partly to avoid repetition of the situation in 1942 when there were not enough competent teams to control malaria in overseas forces, partly to deal with infections introduced into the United States, and partly to combat certain endemic diseases, notably murine typhus, sylvatic plague and insect-borne virus infections, which are progressively infiltrating and entrenching themselves in new sections of the United States. *Charles Wilcocks.*

RHODESIA, SOUTHERN. **Report of the National Health Services Inquiry Commission 1945**. pp. iv+124. 1946. Salisbury : Rhodesian Printing and Publishing Co., Ltd.

The Commission consisted of Professor C. F. M. Saint (Chairman) and four members ; the terms of reference covered enquiry into the following matters :—

" (i) The provision of an organised National Health Service in conformity with the modern conception of 'Health' which will ensure adequate medical, dental, nursing and hospital services for all sections of the people of Southern Rhodesia.

" (ii) The administrative, legislative and financial measures which would be necessary in order to provide Southern Rhodesia with such a National Health Service."

It would not be possible within a short space to give even the headings of this comprehensive report, and those who are interested in the subject should consult the original. The Commission have made recommendations for the future development of the medical services which, if carried into effect, should exercise a profound effect on the health of all sections of the community.

They have evidently been influenced by the schemes prepared in the Union of South Africa and by the Foréami service of the Belgian Congo. There is much evidence of the need to integrate the health services with those relating to agriculture and animal husbandry.

This report is one more sign of the need felt, throughout Africa, for a revision of existing organizations in relation to health ; there is little doubt that, in the near future, that continent as a whole will be much better served than hitherto.

Charles Wilcocks.

MOTTOULLE, L. Quelques statistiques médicales et démographiques concernant la main-d'oeuvre indigène de l'Union Minière du Haut-Katanga. [**Some Medical and Demographic Statistics relating to the Native Craftsmen in the Upper Katanga Mines.**] 1925–1944. *Ann. Soc. Belge de Méd. Trop.* 1946, June 30, v. 26, Nos. 1/2, 81–5.

MANSON, J. M. S. **The Maldive Islands and their Inhabitants.** *J. Roy. Nav. Med. Serv.* 1946, Apr., v. 32, No. 2, 105–12, 6 figs. [10 refs.]

There is little available information on health conditions in the Maldive Islands, and the present communication is therefore of particular interest. The islands comprise a group of coral atolls and individual islands, 400 miles south-west of Ceylon. The people are of Arabian, Indian and Ceylonese origin, and are Moslems. The population of the southern islands numbers 14,500, and as a rule the people are mostly concentrated in one village on each island. The houses are primitive, with roofs of iron, thatch or tiles ; the floors are of coral sand, which is removed and replaced frequently. Beside each house is at least one well, for drinking water, but there is also usually a second, for washing. Clothing is simple ; the ordinary people do not wear shoes or sandals, which are the prerogative of royalty. There is no caste system, but social distinctions exist ; the Sultan is the ruler, and his Cabinet consists of men closely related to him.

Diet consists chiefly of rice, which is all imported, and of the products of the rather poor soil—coconuts, papayas, bananas, bread-fruit, sweet potatoes, yams, pumpkins, limes and mangoes—fowls (with their eggs) and fish. Toddy is made from the young shoots of coconut trees, but is consumed fresh ; no alcoholic liquor is allowed. Education is slight, and the people do not approve of new ideas ; it is forbidden to introduce modern inventions. Modern medical attention is not available. The commonest malady is chronic ulceration of the legs, but malaria, filariasis, elephantiasis and anaemia are prevalent. Amoebiasis, bacillary dysentery, tuberculosis, bronchitis, asthma, poliomyelitis and leprosy are also mentioned.

During the war scrub typhus became of great importance ; it is endemic in the Maldive Islands, and caused much trouble among the British forces, who were stationed there. The papers by HAY [this *Bulletin*, 1945, v. 42, 109] and ZAIR [*ibid.*, 111] refer to the Maldives ; there were many cases of scrub typhus in men of the Royal Navy, and though the case-mortality rate was not high, the morbidity rate was considerable. The larval mites concerned in transmission were probably *Trombicula deliensis*, but proof of this could not be obtained. In the indigenous people there is no evidence that scrub typhus is common, but infections are probably acquired in childhood.

The author discusses malaria, which is prevalent. [He states that the vector is *Anopheles fasciolatus*. No anopheline species of that name is known to the reviewer, nor is *fasciolatus*, so far as can be ascertained, a synonym of any other species. COVELL (*J. Malaria Inst. of India*, 1944, v. 5, 424) notes that *Anopheles tessellatus* is the only anopheline so far recorded from the Maldive Islands, and that it is responsible for the transmission of malaria there ; it breeds in shallow, freshwater wells. No doubt *A. tessellatus* is the species meant.]

Charles Wilcocks.

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Am.	signifies	Amoebiasis and Intestinal Protozoal Infections.
Bart.	,,	Bartonellosis.
Bl.	,,	Blackwater.
B.R.	,,	Book Review.
Chl.	,,	Cholera.
Def. Dis.	,,	Deficiency Diseases including Epidemic Dropsy.
Den.	,,	Dengue, Sandfly and Allied Fevers.
Der.	,,	Dermatology and Fungous Diseases.
Dys.	,,	Dysentery (Bacillary and Unclassed).
Ent.	,,	General Entomology.
Haem.	,,	Haematology.
Heat Str.	,,	Heat Stroke and Allied Conditions.
Hel.	,,	Helminthiasis.
Lab.	,,	Laboratory Procedures.
Leish.	signifies	Leishmaniasis.
Lep.	,,	Leprosy.
Mal.	,,	Malaria.
Misc. Dis.	,,	Miscellaneous Diseases.
Oph.	,,	Tropical Ophthalmology.
Pl.	,,	Plague.
Prot.	,,	General Protozology.
Rab.	,,	Rabies.
R.F.	,,	Relapsing Fever and other Spirochaetoses.
Reports, etc.	,,	Reports, Surveys and Miscellaneous Papers.
Sp.	,,	Sprue.
Tryp.	,,	Trypanosomiasis.
Typh.	,,	Fevers of the Typhus Group.
Ulc.	,,	Tropical Ulcer.
Vms.	,,	Venoms and Antivenenes.
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Ys.	,,	Yaws.

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Alf, S., 566 (Hel.)
Alfonso, J., with Sotolonga & Del Valle Alemán, (50) (Hel.)
Alfonso y Armenteros, J., with de Castro Palomino, 563 (Lep.)
Allan, P., 498 (Reports, etc.)
Allen, A. C. & Spitz, S., 25 (Typh.)
Allen, G. M., 689 (B.R.)
—— & Goodale, R. H., 856 (Der.)
Allen, R. F., with Stubenford, 1050 (Hel.)
Allibone, E. C. & Baar, H. S., 1061 (Def. Dis.)
Alpatov, V. V., Nastukova, O. K. & Khartulari, E. M., 69 (Ent.)
Alpins, O., with Schmitt & Chambers, 482 (Der.)
Alsever, W. D., with Cormia, 1167 (Der.)
Alston, W. F., with Greaves & Gezon, 445 (R.F.)
Altmann, A., 372, 1165 (Haem.)
Alves, W., 752 (Hel.)
—— & Blair, D. M., 344 (Hel.)
Alving, A. S., with Heidelberger, Mayer, Craige, Jones, Pullman & Whorton, 1013 (Mal.)
Amaral, D. F., with Pessoa, 43 (Am.)
Amberson, J. M., 440 (Chl.), 1046 (Hel.)

Amendola, F., 225 (Lep.)
American Association for the Advancement of Science, 881 (B.R.)
Amin, N., Mahfouz, M. & Sherif, M. A. F., 43 (Am.)
Ananian, S. A., Chubkova, A. I., Azizian, A. A. & Kocharian, A. A., 13 (Mal.)
Andersen, D. A., (229) (Hel.)
Andersen, H., 971 (B.R.)
Anderson, B. G., 531 (Mal.)
Anderson, E. S., 1144 (R.F.)
Anderson, G. W., with Simmons, Whayne, Horack & collaborators, 86 (B.R.)
Anderson, N., with Moore, Kessel, Simonsen, Marmorston, Llewellyn, Kaplan, Golden & Jaffe, (650) (Dys.)
Anderson, W. L. & Wing, W. M., 326 (Typh.)
Anderson, W. N., with Fowweather, 1064 (Sp.)
Andrade, S. O., with Rocha e Silva & Porto, 1052 (Hel.)
d'Andretta, M. A. V. & d'Andretta, C., Jr., (1195) (Ent.)
Andrew, R., 121, 549 (Typh.), 522 (Mal.)
Andrewes, C. H., King, H. & Walker, J., 427, 728 (Typh.)
Andrews, J. M., 1200 (Reports, etc.)
Anigstein, L., 1031 (Typh.)
—— & Bader, M. N., 1036 (Typh.)
Ann. Trop. Med. & Parasit., 99 *bis*, (394), 905 (Mal.)
Ansari, M. N. 1127 (Leish.)
Antonio Echandi, G., R. with Sutliff, (1057) (Hel.)
Antonio Quiroz, J., with Puig Solanes & Fontes, 764 (Hel.)
Antunes, W. & Castro, A., 643 (Y. F.)
Arantes, J. B., Karmann, G. & Bier, O. G., (670) (Vms.)
Arbogast, J. L., with Cox, 1187 (Misc. Dis.)
d'Arcangelo, D., (1077) (Misc. Dis.)
——, with Buffa, (1077) (Misc. Dis.)
Arch. Inst. Pasteur d'Algérie, (13) (Mal.)
Archetti, I., (832) (Leish.)
Argüello Pitt, L. & Alberto Consigli, C., (852) (Lep.)
—— & Conejos, M., 456, 849 (Lep.)
——, with Garzón, 849 (Lep.)
Armbrust, C. A., Jr., with Irons, 550 (Typh.)
Armstrong, T. G., 425 (Leish.)
Arnold, E. H., Ferguson, F. F. & Upholt, W. M., 793 (Ent.)
——, with Upholt, Gaines & Simmons, 794 (Ent.)
Arnold, H. L., Jr., 751 (Lep.)
—— & Tilden, I. L., 452 (Lep.)
Arthur, J. H., with Afridi, 204 (Mal.)
Arouck, R., with Damasceno & Causey, (1195) (Ent.)
Ash, J. E. & Spitz, S., 691 (B.R.)
Ashburn, L. L., Perrin, T. L., Brady, F. J. & Lawton, A. H., 662 (Hel.)
Ashley, P., 1188 (Misc. Dis.)
Atchley, J., Bacon, R., Curran, G. & David, K., 160 (Lab.)
Aubin, H., 322 (Typh.)
Audibert, Y., with Sautet, 789 (Ent.)
Augustine, D. L., 1025 (Tryp.)
Augustine & Lherisson, C., 570 (Hel.)
Ault, C. N., (798) (Ent.)
Austin, R. S., with Blankenhorn, Vilter & Scheinker, 945 (Def. Dis.)
Australian Army Medical Research Unit, Cairns, Queensland, 527, 628 (Mal.)
Australian Military Forces, 516 *bis*. (Mal.)
Austrian, R., with Whitehill, 450 (Ys.)
Austrian, R. C., with Mackie, Davis, Fuller, Knapp, Steinacker, Stager, Traub, Jellison, Millspaugh, Bell, Kohls, Wie Hsi & Girsham, 917, (Typh.)
Avera, J. W., Yow, E. M., Harrell, G. T. & Fowler, E. B., 765 (Hel.)
Avery, J. L., 785 (Misc. Dis.), 1046 (Hel.)
Awni, A. Y., with Halawani & Nor-El-Din, 1142 (Am.)
Axtmayer, J. H., with Krakower & Hoffman, 227 (Hel.)
Ayayao, C. D., with Fernando & Cruz, 1181 (Oph.)
de Azevedo, J. F., Cambournac, F. J. C. & Pinto, M. R., (716), 828, (829) (Tryp.)
Azizian, A. A., with Ananian, Chubkova & Kocharian, 13 (Mal.)
Azulay, R. D., with Convit, Bermudez & Salgado, 457 (Lep.)

B.

Baar, H. S., with Allibone, 1061 (Def. Dis.)
Babin, F. & Dulaney, A. D., 297 (Mal.)
Babione, R. N., 576 (Hel.)
Bablet, J., 263 (B. R.)
—— & Giroud, P., (322) (Typh.)
Babouot, P., with Martin, Le Roy, Sureau & Bourcart, 464 (Hel.)
Bacon, R., with Atchley, Curran & David, 160 (Lab.)
Badenski, A., with Ciuca, Mesrobeanu, Franke & Munteanu, (210) (Typh.)
——, with ——, ——, Munteanu & Paraschivescu, (210) (Typh.)
Bader, M. N., with Anigstein, 1036 (Typh.)
Badhwar, R. L., Nayar, S. L. & Chopra, I. C., 672 (Der.)
Badir, G., 1154 (Hel.)
Bagby, J. W., 149 (Der.)
Bagchi, A. K., with Majumder & Ghosh, 441 (Dys.)
Baggenstoss, A. H., with Bayley & Lindberg, (697) (Misc. Dis.)
Bailly, J., 184 (Rab.)
Baker, A. B., with Chalgren, (694) (Misc. Dis.)
Baker, M., with Brackett & Waletzky, 411, 713 (Mal.)
Ballif, L., with Ciuca & Chelarescu, 292 (Mal.)
——, ——, —— & Cristescu, 290 (Mal,)
Ballou, A. N., with Adams, 866 (Lab.)
Baltazard, M., with Blanc, 1038 (Pl.)
Bancroft, H., with Doull, Guinto & Rodriguez, 453 (Lep.)
——, Guinto, R. S., Rodriguez, J. N. & Marques, A. P., 454 (Lep.)
Bang, F., Ferguson, M. S., Hairston, N. G. & Graham, O. H., 350 (Hel.)

Bang, F. B., with Trager & Hairston, 821 (Mal.)
Baranovskaya, S. A., 193 (Rab.)
Barber, H. J., Major, F. W. & Wragg, W. R., (1011) (Mal.)
—— & Wragg, W. R., (1011) (Mal.)
Bargmann, E. E., with Buckland, Dudgeon, Edward, Henderson-Begg, MacCullum, Niven, Rowlands, van den Ende, Curtis & Shepherd, 436 (Typh.)
Barlow, C. H. & Abdel Azim, M., 753 (Hel.)
Barnes, R. C., with Felton & Wilson, 308 (Mal.)
Barnett, L., 352 (Hel.)
Barreda, E. A., with Fairchild, 680 (Ent.)
Barreto, A. L. de B. & Pondé, A., (424) (Tryp.)
Barretto, M. P., with Pessoa, 1030, 1127, 1128 (Leish.)
Barros, J. de R., 1156 (Hel.)
Bartlett, A. G., with Cherry, 778 (Der.)
Basford, F. R., Curd, F. H. S. & Rose, F. L., (1110) (Mal.)
Baskind, E., with Merskey, 952 (Haem.)
Basombrio, G., with Mom, 455 (Lep.)
Basset, with Montel, (342) (Lep.)
Basso, G., with Mazza & Basso, 720, 909 (Tryp.)
Basso, R., with Mazza & Basso, 720, 909 (Tryp.)
Basu, U. P., with Bose & Sen Gupta, 219 (Dys.)
Bates, M., 383 (Ent.)
——, with Gast, 644 (Y.F.)
—— & Roca-Garcia, M., 32, 328 (Y.F.)
Baxter, S., (594) (Heat Str.)
Bayley, E. C., Lindberg, D. O. N. & Baggenstoss, A. H., (597) (Misc. Dis.)
Bayliss, M., with Nelson, 1046 (Hel.)
Beadle, L. C., 1190 (Ent.)
Beadry, M., with Berger & Gaumond, 150 (Der.)
Beall, J. C., 639 (Typh.)
Beament, J. W. L., (384), (598) (Ent.)
Beard, J. W., with Cooper & Rein, 1027 (Leish.)
Beaver, P. C., with McMullen, 138 (Hel.)
Bechelli, L. M., Keil, H. & Rotberg, A., 225 (Lep.)
Becker, B. J. P., 1076 (Misc. Dis.)
Becker, F. T., with Kaplan & Read, 1110 (Mal.)
Beeson, P. B., with Miller, 732 (Typh.)
Beet, E. A., 774 (Haem.)
Behm, A. W. & Hayman, J. M., Jr., 762 (Hel.)
Beklemishev, V. N., Shlenova, M. F. & Orlov, P. M., 1112 (Mal.)
Belkin, J. N., (383) (Ent.)
——, Knight, K. L. & Rozeboom, L. E., 6 (Mal.)
Bell, E. J., with Blake, Maxcy, Sadusk & Kohls, 327 (Typh.)
——, with Mackie, Davis, Fuller, Knapp, Steinacker, Stager, Traub, Jellison, Millspaugh, Austrian, Kohls, Wei Hsi & Girsham, 917 (Typh.)
—— & Plotz, H., 434 (Typh.)
Bell, F. R. & Jones, E. R., (1123) (Tryp.)
Bell, J. E., Jr. & Boone, J. A., 377 (Vms.)
Beltrán, E., (620) (Mal.)
——, with Leon & Larenas M., 1017 (Mal.)
Benda, R. & Franchel, F., 1198 (Lab.)
Bengtson, I. A., 20, 551 (Typh.)
——, Topping, N. H. & Henderson, R. G., 19 (Typh.)
——, with —— & ——, 20, 21 (Typh.)
——, with —— & Shear, 18 (Typh.)
Bennett, B. L., with Plotz & Reagan, 920 (Typh.)
——, with —— & Wertman, 725 (Typh.)
Bennett, R. E., with Lawrence, 36 (Dys.)
Bensimon, T., with Picard, 42 (Am.)
Bent. M. J., Turner, E. L., Holloway, G. D. & Cuff, J. R., 253 (Misc. Dis.)
Ber, M., with Adler & Tchernomoretz, 114 (Leish.)
Berberian, D. A., 18 (Leish.), 653 (Am.), 939 (Hel.), 1105 (Mal.)
Bercovitz, Z. T., 406 (Mal.)
Berg, W. N., 242 (Haem.)
Berge, C., with Le Chuiton, 915 (Typh.)
Berger, E. & Brzezinski, S., 543 (Typh.)
Berger, L., Beadry, M. & Gaumond, E., 150 (Der.)
Bergeret, C., with Despujols, Calmet & Rouvier, (253) (Misc. Dis.)
Berkowitz, S., 157 (Misc. Dis.)
Berlin, C., 856 (Der.)
Berman, R. L., with Friedheim, 1124 (Tryp.)
Bermudez, D., with Convit, Azulay & Salgado, 457 (Lep.)
Bernard, L., with Cavaillon, Boyer & Delecourt, 544 (Typh.)
Berry, J. F., with Zarafonetis & Ingraham, 746 (R.F.)
Berry, M. G., with Johnson, 351 (Hel.)
Berteaux, M., with Vincke, Parent & de Meulenaere, 291 (Mal.)
Bertram, D. S., 1197 (Ent.)
——, Unsworth, K. & Gordon. R. M., 1197 (Ent.)
Bethea, W. R., Jr., with Brown & Stifler, 939 (Hel.)
Bevier, G., 1136 (Y.F.)
Bhatnagar, S. S. & Shrivastava, D. L., 840 (Pl.)
Bhattacharjya, B. P. & Guha, G. S., 1178 (Oph.)
Bhattacharya, K. P., with Rudra, 787 (Misc. Dis.)
Biaggi, N., with Oliver González & Rivera León, 347 (Hel.)
Bianchi, A. E., 63 (Der.)
Bianco, A. A., with Smith, Stormont & Evans, (1157) (Hel.)
Bien, W. N., 363 (Def. Dis.)
Bier, O. G., (670) (Vms.)
——, with Arantes & Karmann, (670) (Vms.)
Bierbaum, O. S., with Moore, Welch & Wright, 477 (Haem.)
Bierring, W. L., with Woofter & Dick, 372 (Haem.)
Biesheuvel, S., 507 (B.R.)
Biggam, A. G., 783 (Misc. Dis.)
Bijlmer, J., (356) (Hel.)
Billings, J. J., with Hall, 443 (Am.)

Binford, C. H. & Lane, J. D., 487 (Der.)
Binson, G., 217 (Chl.)
Birnkrant, W. B., Greenberg, M. & Most, H., 221 (Am.)
Birrell, N. V., 999 (Mal.)
Bishay, A., (706) (Mal.)
Bishop, A. & Gilchrist, B. M., 710 (Mal.)
Black, D. A. K. & Fourman, P., 474 (Sp.)
——, Fourman, L. P. R. & Trinder, P., 667 (Sp.)
——, —— & Trinder, P., 1161 (Sp.)
Black, K. O., 48 (Hel.)
Black, R. H., 815 (Mal.)
Blackith, R. E., with Page & Stringer, 600 (Ent.)
Blacklock, M. G., with Moyne, Stubbs, Crowdy, Citrine, Mackinnon, Engledow & Henderson, 70 (Reports, etc.)
Blagoveschensky, D., Bregetova, N. & Monchadsky, A., (160) (Ent.)
Blair, D. M., with Alves, 344 (Hel.)
Blake, F. G., Maxcy, K. F., Sadusk, J. F., Jr., Kohls, G. M. & Bell, E. J., 327 (Typh.)
Blakeslee, E. B., (494) (Ent.)
——, with Bruce, (1194) (Ent.)
Blanc, G. & Baltazard, 1038 (Pl.)
Blanchard, M., 604 (Reports, etc.)
Blanco, J. F., 851 (Lep.)
Bland, J. O. W., 1172, (1184) *bis* (Oph.)
—— & Wilson, R. P., 1174 (Oph.)
Blankenhorn, M. A., Vilter, C. F., Scheinker, I. M. & Austin, R. S., 945 (Def. Dis.)
Blass, J., 742 (Chl.)
Bloch, E. H., with Knisely & Eliot, 631 (Mal.)
——, with ——, Stratman-Thomas & Eliot, 1113 (Mal.)
Bloom, W., with Taliaferro, 630 (Mal.)
Bloss, J. F. E., 656 (Lep.)
Blum, H. F., 152 (Heat Str.)
Bobrinskoy, N., Kuznetzov, B. & Kuzyakin, A., 268 (B.R.)
Böe, J., 443 (Am.)
Boehnhardt, H., 128 (Den.).
Bohart, G. E., with Weathersbee, 291 (Mal.)
Bok, R., 748 (R.F.)
Boland, E. W., Headley, N. E. & Hench, P. S., 597 (Misc. Dis.)
Bonatti, A. A. & Olmos Castro, N., 850 (Lep.)
——, with ——, 849 (Lep.)
Bonnet, R., (232) (Hel.)
Bookless, A. S. & Naftalin, J. M., 199 (Mal.)
Boone, J. A., with Bell, 377 (Vms.)
Boquet, P., 148, 1068 (Vms.)
Boragina, R. C., with Taiana & Niño, 487 (Der.)
Borden, D. L., Waddill, J. F. & Grier, G. S., 151 (Heat Str.)
Borges-Fortes, A., 316 (Tryp.)
Borjeix, L., with Le Gac, (327) (Typh.)
v. Bormann & Preuss, 324 (Typh.)
Boscardi, F., (852) (Hel.)
Bose, A. N. & Ghosh, J. K., 36 (Dys.)
——, —— & Rakshit, P. C., 928 (Dys.)
——, Ghosh, T. N., Mitra, S. N. & Datta, S., 1028 (Leish.)
——, Sen Gupta, P. N. & Basu, U. P., 219 (Dys.)
Boshell-Manrique, J., with Kumm & Osborno-Mesa, 599 (Ent.)
Bound, J. P., with Keele, 368 (Sp.)
Bourcart, N., with Martin, Le Roy, Sureau & Babouot, 464 (Hel.)
Bourgain, M., (318 *bis*) (Leish.)
—— with Pirot, 336, 337 *bis* (R.F.)
Bovarnick, M. R., Lindsay, A. & Hellerman, L., 824, 825 (Mal.)
Boventer, K. & Fischer, R., 546 (Typh.)
Bovet, D., with Funke & Montézin, 713 (Mal.)
——, with Lwoff & Funke, 313 (Tryp.)
Bow, M. R. & Brown, J. H., 327 (Typh.)
Boyd, J. S. K., 783 (Misc. Dis.)
Boyd, M. F., 289 (Mal.), (688) (Reports, etc.)
——, & Kitchen, S. F., 304, 1003 (Mal.)
Boyd, W. S., Stubbs, T. H. & Weinstein, P. P., 1200 (Reports, etc.)
Boyer, with Cavaillon, Bernard & Delecourt, 544 (Typh.)
Bozicevich, J., Hoyem, H. M. & Walston, V. M., 798 (Lab.)
Bozman, C. A., 968 (Reports, etc.)
Brackett, S. & Hughes, C. O., 15 (Mal.)
—— Waletzky, E. & Baker, M., 411, 713 (Mal.)
Bradley, G. H. & Fritz, R. F., 198 (Mal.)
Brady, F. J., with Ashburn, Perrin & Lawton, 662 (Hel.)
——, with Lawton, Ness & Haskins, 55 (Hel.)
Brasil, A. 908 (Typh.)
Bratton, A. C., Jr., 408, 413 (Mal.)
Braun, E., with Dole & Emerson, 624 (Mal.)
Brazzaville [A. E. F.], 190 (Rab.)
Brecher, G., 676 (Ulc.)
Bregetova, N., with Blagoveschensky & Monchadsky, (160) (Ent.)
Brennan, J. M., 122 (Typh.)
Brenner, S., with Gillman & Gillman, 365, 366 (Def. Dis.)
Breslow, D. S., Walker, H. G., Yost, R. S., Shivers, J. C. & Hauser, C. R., (406) (Mal.)
Breslow, L., with Johnson & Martin, 645 (Den.)
Brierly, W. B., 620, 621 (Mal.)
de Briey, P., 605 (Reports, etc.)
Briggs, G. D. S., (905) (Mal.)
Brinton, D., 594 (Misc. Dis.)
Brisou, J., with Magrou, 926 (Pl.)
British Guiana, 286 (Mal.), 1136 (Y.F.)
Brodie, J., Cook, R. P., Drysdale, C. F. & McIntosh, D. G., 841 (Dys.)
Brody, J., 1036 (Typh.)
Brothers, W. C., with Wisecup & Eide, 864 (Ent.)
——, with ——, —— & Deonier, 1078 (Ent.)
Brousseau, D., with Mayer, 1125 (Tryp.)
Brown, A. G., 650 (Am.)
Brown, H. W., 939 (Hel.)
——, with Williams, 663 (Hel.)
Brown, J. H., with Bow, 327 (Typh.)
Brown, R. L., 684 (Lab.)
Brown, T. McP., Stifler, W. C., Jr. & Bethea, W. R., Jr., 939.
Browne, G. St. J. O., 883 (B.R.)
Browning, C. H., Calver, K. M., Leckie, M. W. & Walls, L. P., 633 (Tryp.)
Browning, J. S., Raphael, M., Klein, E. F. & Coblenz, A., 548 (Typh.)

Brooks, A. G., with Ahuja, 478 (Vms.)
Bruce, W. G. & Blakeslee, E. B., (1194) (Ent.)
Brückmann, G. & Wertheimer, E., 206 (Bl.)
Brueck, J. W., with Burns & Moss, 486 (Der.)
Brueckmann, G. & Wertheimer, E., 334 (Am.)
Bruetsch, W. L., (13), (298) (Mal.)
Brug, S. L., 288 (Mal.), 321 (Typh.), 1041 (Am.)
—— & Vos, J. J. T., 382 (Prot.)
Brumpt, E., 4, (907) (Mal.)
Brumpt, L. C. & Maclouf, A. C., 835 (Typh.)
Brun, Mlle., with Montel & Marliangeas, Mlle., 454 (Lep.)
Brunsting, H. A., with Gordon, Marble, Engstrom & Lippincott, 1007 (Mal.)
Bruwer, T., with Palley, 1083 (Reports, etc.)
Brzezinski, S., with Berger, 543 (Typh.)
Buchans, R. A., 231 (Hel.)
Buckland, F. E., Dudgeon, A., Edward, D. G. ff., Henderson-Begg, A., MacCallum, F. O., Niven, J. S. F., Rowlands, I. W. & Van den Ende, M., with Bargmann, H. E., Curtis, E. E. & Shepherd, M. A., 436 (Typh.)
Buffa, F. & d'Arcangelo, D., (1077) (Misc. Dis.)
Bugher, J. C., 215 (Y.F.)
Bull. Health Organisation (607) (Reports, etc.)
Bull. U.S. Army Med. Dept., 739 (Pl.), 925 (Den.)
Burch, G. E., 593 (Heat Str.)
—— & Winsor, T., (675) (Heat Str.)
——, with ——, 243 (Haem.)
Burgess, R. W., with Young, Stubbs, Moore, Ehrman, Hardman & Ellis, 5 (Mal.)
de Burgh, P., 642 (Typh.)
de Burgh, P. M., 966 (Lab.)
Burke, E., 317 (Leish.)
Burnham, R. C., 525 (Mal.)
Burns, E. L., Moss, E. S. & Brueck, J. W., 486 (Der.)
Burrell, R. W., with Deonier, 410 (Mal.)
——, with ——, Maple & Cochran, 101 (Mal.)
——, with Eide & Deonier, 600 (Ent.)
——, with Jones, Deonier & Knipling, 530 (Mal.)
——, with Wisecup & Deonier, 530 (Mal.)
Burroughs, A. M., with Hawking, (940) (Hel.)
Burrows, R. B., (227) (Hel.)
Burtt, E., 827, 828, 1121 *bis* (Tryp.)
Bushland, R. C., 922, 923 (Typh.), 1196 (Ent.)
Büssow, H., 297 (Mal.)
Bustamante, M. E., Varela, G. & Ortiz Mariotte, C., 1134 (Typh.)
Butt, E. M. & Hoffman, A. M., 63 (Der.)
Butts, D. C. A., 533 (Bl.)
Buxton, P. A., 305 (Mal.)
Bystrov, P. V., 294 (Mal.)

C.

Cadenat, J., with Parrot & Mornet, (160), (602) (Ent.)
Cairo, 753 (Hel.)
Calamet, L., with Violle, (730) (Typh.)
Caldeira, R. da G., 1153 (Lep.)
Caldwell, J. D., 1104 (Mal.)
Caldwell, M. H., with Spies, Vilter & Koch, 375 (Haem.)
California Mosquito Control Association, 1079 (Ent.)
Callahan, W. P., Jr., 254 (Prot.)
Calmet, L., with Despujols, Bergeret & Rouvier, (253) (Misc. Dis.)
Calver, K. M., with Browning, Leckie & Walls, 633 (Tryp.)
Calvery, H. O., with Fitzhugh, Nelson & Glassman, 525 (Mal.)
Cambournac, F. J. C., with de Azevedo & Pinto, (716), 828, (829) (Tryp.)
—— & Simões, J. M. P., 95, 306 (Mal.)
Cameron, G. R., 835 (Typh.)
Cameron, T. W. M., 679 (Ent.), 1048 (Hel.)
Campana, Y., with Desportes, (746) (R.F.)
Campau, E. J., with Kartman, Newcomb & Morrison, 716 (Tryp.)
Campbell, D. H. & Cherkin, A., 555 (Chl.)
Campos, J. A., 834 (Leish.)
Campos, N. S. & Souza, P. R., 47 (Lep.)
Canaan, T., 319 (Leish.), 340 (Lep.)
Cano, C., with León, 731 (Typh.)
Cantrell, W., with Coulston & Huff, 205 (Mal.)
——, with Kelsey, Oldham & Geiling, (625) (Mal.)
Carini, A., (1123) (Tryp.)
—— & Springer, L., (1170) (Der.)
Carpanelli, J. B., with Alecha & Ferreira, (136) (Am.)
Carrico, C. C., with Lippincott, Ellerbrook, Hesselbrock & Marble, 1009 (Mal.)
Carrión, A. L. & Knott, J., 246 (Der.)
Carrol, D. G., 1047 (Hel.)
Carruthers, L. B., 950 (Sp.)
Carter, H. F., (1000) (Mal.)
—— & d'Abrera, V. St. E., 683 (Ent.)
Carver, R. K., with Wharton, 1079 (Ent.)
Casals, J., 191 (Rab.)
Case, R. A. M., 835 (Typh.), 1164 (Haem.)
Cassinelli, J. F., with Lasnier, 567 (Hel.)
Castellanos, L., with Woodruff, (488) (Der.)
Caster, W. O., with Mickelsen & Keys, 1061 (Def. Dis.)
Castillo, R. L., 262 (B.R.)
Castle, W. B., with Watson, 1067 (Haem.)
Castro, A., with Antunes, 643 (Y.F.)
de Castro Ferreira, L., with Laemmert, 33 (Y.F.)
de Castro Palomino, J. & Alfonso y Armenteros, J., 563 (Lep.)
Catanei, A., 780 (Der.)
—— & Kervran, P., 247 (Der.)
Cateno, C. F., with Ellerbrook, Lippincott, Gordon & Marble, 626 (Mal.)
Cattan, R., Corcos, A. & Cohen, H., 337 (R.F.)
Caubet, P., 312 (Tryp.)
——, with Roubaud, 418 (Tryp.)
——, with Stefanopoulo & Duvolon, Mlle., 417, 420 (Tryp.)
Causey, O. R., with Damasceno & Arouck, (1195) (Ent.)
——, Deane, L. M. & Deane, M. P., (199) (Mal.)
—— & Mello, G. B., 287 (Mal.)

Cavaillon, A., Bernard, L., Boyer & Delecourt, 544 (Typh.)
Cawston, F. G., (1190) (Misc. Dis.)
Ceccaldi, J., 190 (Rab.)
Cekada, E. B., with Rifkin, Zarrow, Henderson & Whitehead, 188 (Rab.)
Cerboni, E. A., Pecoraro, V. & Rodolfo Mercau, A., 847 (Lep.)
Cerqueira, G. de C., with Lima, 47, 226 (Lep.)
Cerri, B., 749 (Lep.)
Cerruti, H., 1129 (Leish.)
Chabeuf, M. & Mbappe, 1122 (Mal).
Chakravarti, H., with Das Gupta & Lowe, 298 (Mal.)
Chakravarty, K. C., 833 (Leish.)
Chakravarty, N. K., with Sen Gupta, 833, 834 (Leish.)
Chalgren, W. S. & Baker, A. B., (594) (Misc. Dis.)
Chalke, H. D., 835, 911 (Typh.)
Chambers, G., with Schmitt & Alpins, 482 (Der.)
Chandler, A. C., 1085 (B.R.)
Chandy, P. J., 656 (Lep.)
Chapin, S. E., 844 (Am.)
Chardome, M., with Peel, 1159 (Hel.)
Charters, A. D., 224 (R.F.), (474) (Def. Dis.) 551 (Typh.)
——, with Manson-Bahr, 1186 (Misc. Dis.)
Chatterjea, J. B., with Das Gupta & Ganguli, 1066 (Haem.)
Chatterjee, B. C., with Gupta, Paul & Ghose, 130 (Chl.)
Chatterjee, H. N., 555 (Chl.), 722 (Leish.)
Chatterjee, M., with Gupta & Panja, 1138 (Pl.)
Chaudhri, J. R., with Yacob, 129, 649 (Chl.)
Chaudhuri, R. N. & Rai Chaudhuri, M. N., 843, (844) (Am.)
Cheever, F. S., 649 (Dys.)
Chelarescu, M., with Ciuca & Ballif, 292 (Mal.)
——, with ——, —— & Cristescu, 290 (Mal.)
Chen, G. & Geiling, E. M. K., 315 (Tryp.)
Chen, K. T., Tang, I. L. & Wang, M. C., (97) (Mal.)
Chen, S. M., with Pang, Zia & Feng, 326 (Typh.)
Cheney, G., with Noe & Greene, 521 (Mal.)
Cherkin, A., with Campbell, 555 (Chl.)
Chermock, R. L. & Muller, H. E., 968 (Lab.)
Cherry, C. B. & Bartlett, A. G., 778 (Der.)
Chesterman, C. C., 835 (Typh.)
Ch'i Ho, (1000) (Mal.)
Chiao, S. M., 930 (R.F.)
Chippaux, C. & Chippaux-Mathis, J., 788 (Prot.)
Chippaux-Mathis, J., with Chippaux, 788 (Prot.)
Chopra, I. C., with Badhwar & Nayar, 672 (Der.)
——, with Chopra, 304 (Mal.)
Chopra, R. N. & Chopra, I. C., 304 (Mal.)
Chorine, V., 341, 459, 564, (Lep.)
Chorley, J. K., 422 (Tryp.)
Christophers, R., 684 (Reports, etc.)
Chubkova, A. I., with Ananian, Azizian & Kocharian, 13 (Mal.)
Chung, H. L., 321 (Typh.)
Churchill, M. H., 473 (Def. Dis.), 588 (Oph.)
Chwatt, L. J., 5 (Mal.)
Ciaravino, E., with Sofia, 37 (Am.)
Cifuentes, O., with Macchiavello, 23 (Typh.)
——, with —— & Ovalle, 1032, 1033 (Typh.)
Citrine, W., with Moyne, Stubbs, Crowdy, Mackinnon, Blacklock, Engledow & Henderson, 70 (Reports, etc.)
Ciuca, M., Ballif, L. & Chelarescu, M., 292 (Mal.)
——, ——, —— & Cristescu, A., 290 (Mal.)
——, with Ionesco-Mihaiesti, 321 (Typh.)
—— & Mesrobeanu, L., (210) (Typh.)
——, ——, Badenski, A., Franke, M. & Munteanu, G., (210) (Typh.)
——, ——, ——, Munteanu, G. & Paraschivescu, N., (210) (Typh.)
Clancy, C. F. & Wolfe, D. M., 641 (Typh.)
——, with ——, Vanderscheer & Cox, 735 (Typh.)
Clapham, P. A., (233) (Hel.)
Clapp, J. M., with Fay & Simmons, 790 (Ent.)
Clark, E. M., with Craigie, Watson & Malcomson, 914 (Typh.)
Clark, J. H., with English, Shepherd, Marson, Krapcho & Roblin, (905) (Mal.)
Clark, R. H. P., 1132 (Typh.)
—— & Dutta, D. K., 559 (Am.)
Clarke, C. A. & Sneddon, I. B., 944, 1059 (Def. Dis.)
Clarke, J. D., 1024 (Tryp.)
Claus, G., 115 (Typh.)
Clavero, G. & Gallardo, F. P., (117) (Typh.)
Cleland, J. B., (42) (Am.)
Clemo, G. R., Cocker, W. & Hornsby, S., (1054) (Hel.)
Cline, J. K., with Spies, Vilter & Frommeyer, 772 (Haem.)
Cluver, E. H., Jokl, E. & Rorich, P. R., (1084) (Reports etc.)
Cluver, F. W. P., 1111 (Mal.)
Coates, W. A., with Heidelberger & Mayer, 1013 (Mal.)
Coatney, G. R., Cooper, W. C. & Trembley, H. L. 629 (Mal.)
Coblenz, A., with Browning, Raphael & Klein, 548 (Typh.)
Cochran, J. H., with Deonier, Burrell & Maple, 101 (Mal.)
Cochrane, R. G., 1146 (Lep.)
Cockburn, C., (566) (Hel.)
Cocker, W., with Clemo & Hornsby, (1054) (Hel.)
Coda, D., (904.) (Mal.)
Codeleoncini, E., 1130, 1131 (Typh.)
——, with d'Ignazio, 732, 1130, 1131 (Typh.), 1042 (R.F.)
Codounis, A., (539) (Leish.)
Coelho, J. T., 1147 (Lep.)
——, with Aleixo, 1150 (Lep.)
Coggeshall, L. T., 295 (Mal.), 852 (Hel.)
——, with Spector & Haviland, 823 (Mal.)
Cogswell, R. C., with Spies, Perry & Frommeyer, 60 (Def. Dis.)
Cohen, H. H., 764 (Hel.)
Cohen, H., with Cattan & Corcos, 337 (R.F.)
Cohen, J., with Rugiero, 326 (Typh.)

Cohen, S. C., Emert, J. T. & Goss, C. C. 595, (Misc. Dis.)
Coito, A. de M. F., with de Meira & Girão, (760) (Hel.)
Colas-Belcour, J., with Roubaud, 383 (Ent.)
Colchester, T. R., (501) (Reports, etc.)
Cole, G., 566 (Hel.)
Cole, H. N. with Luckiesh, Taylor & Sollmann, 675 (Heat Str.)
Cole, L. C., 331 (Pl.)
Collier, H. O. J. & Lourie, E. M., 1126 (Leish.)
Collignon, E., 307 (Mal.)
Collins, D. L., with Glasgow, 924 (Typh.), 962 (Ent.)
Collins, R. K., 999 (Mal.)
Colonial Office, 85, 883 (B.R.)
Combescot de Marsaguet, G., with Le Gac & Seite, 621 (Mal.)
Conejos, M., with Arguello Pitt, 456, 849 (Lep.)
Conge, M., with Nitti & Kauffmann, 749 (R.F.)
Connecticut State Department of Health, 867 (Reports, etc.)
Connell, W. K., 164 (Reports, etc.)
Convit, J., Azulay, R. D., Bermudez, D. & Salgado, P., 457 (Lep.)
Cook, C. D. & Hoffbauer, F. W., 819 (Mal.)
Cook Islands, 75 (Reports, etc.)
Cook, R. P., with Brodie, Drysdale & McIntosh, 841 (Dys.)
Cooke, W. T., Elkes, J. T., Frazer, A. C., Parkes, J. & Peeney, A. L. P., 946 (Sp.)
Cooley, R. A., 1198 (Ent.)
—— & Kohls, G. M., 864 (Ent.)
Coombes, A. E. R., with Rogan, 524 (Mal.)
Cooper, G. R., Rein, C. R. & Beard, J. W., 1027 (Leish.)
Cooper, W. C., with Coatney & Trembley, 629 (Mal.)
Cooperman, J. M., Elvehjem, C. A., McCall, K. B. & Ruegamer, W. R., 772 (Haem.)
——, Ruegamer, W. R. & Elvehjem, C. A., (1067) (Haem.)
Corbett, A. J., with Settle & Pinkerton, 27 (Typh.)
Corbyn, E. N., 76 (Reports, etc.)
Corcos, A., with Cattan & Cohen, 337 (R.F.)
——, with Laigret, 739 (Den.)
Cormia, F. E. & Alsever, W. D., 1167 (Der.)
Cornatzer, W. E., with Kyker & McEwen, 706 (Mal.)
Cornil, L., Poursines, Y. & Moustardier, G., 216 (Pl.)
Corradetti, A., (817) (Mal.)
Corrêa, R. R., (904, 905) (Mal.)
Cottam, C. & Higgins, E., 1014 (Mal.)
Cotter, E., 72 (Reports, etc.)
Cottet, J., with Deschiens, (766) (Hel.)
Cottrell, J. D. & Hayward, G. W., 653 (Am.)
—— & Peddie, J. J. G., 333 (Am.)
Coulston, F., Cantrell, W. & Huff, C. G., 205 (Mal.)
——, with Huff, 1115 (Mal.)
Coupland, R., 1090 (B.R.)
Coutts, W. E., 136 (Am.)
Cowen, D. & Wolf, A., 678 (Prot.)
Cox, C. D. & Arbogast, J. L., 1187 (Misc Dis.)
Cox, H. R., with Irons, Topping & Shepard, 924 (Typh.)
——, with Wolfe, Vanderscheer & Clancy, 735 (Typh.)
Coxon, R. V. & Hayes, W., 406 (Mal.)
Craig, C. F. & Faust, E. C., 506 (B.R.)
Craige, B., with Heidelberger, Mayer, Alving, Jones, Pullman & Whorton, 1013 (Mal.)
Craigie, J., 834 (Typh.)
——, Watson, D. W., Clark, E. M. & Malcomson, M. E., 914 (Typh.)
Crauford-Benson, H. J., 638 (Typh.)
—— & Macleod, J., 638 (Typh.)
Cristescu, A., with Ciuca, Ballif & Chelarescu, 290 (Mal.)
Cropper, C. F. J., 38, 1141 (Am.), 252 (Misc. Dis.)
Croxatto, H., (1069) (Vms.)
Crowdy, R. E., with Moyne, Stubbs, Citrine, Mackinnon, Blacklock, Engledow & Henderson, 70 (Reports, etc.)
Cruickshank, E. K., 1159 (Def. Dis.)
Cruz. E., with Pascale, 546 (Typh.)
Cruz, J. N., with Fernando & Ayuyao, 1181 (Oph.)
Cruz, W. O. & de Mello, R. P., 660 (Hel.)
da Cruz Ferreira, F. S., (796) (Ent.)
—— & de Meira, M. T. V., 863 (Ent.)
Cuff, J. R., with Bent, Turner & Holloway, 253 (Misc. Dis.)
Culbertson, J. T., with Rose & Lipman, 232 (Hel.)
——, —— & Oliver-Gonzalez, J., 54, 354, 763 (Hel.)
Cullinan, E. R., 330 (Den.), 783 (Misc. Dis.), 944 (Def. Dis.)
——, Kekwick, A., Watts, A. S. & Titman, W. L., 942 (Def. Dis.)
da Cunha, A. M., 113, 209 (Leish.)
—— & Guimãraes, F. N., 562 (Ys.)
Curd, F. H. S., with Basford & Rose, (1110) (Mal.)
——, Davey, D. G. & Rose, F. L., 394, 400 (Mal.)
——, Davis, M. I., Owen, E. C., Rose, F. L. & Tuey, G. A. P., (1110) (Mal.)
—— & Rose, F. L., (822 *bis*), (1110) (Mal.)
Curran, G., with Atchley, Bacon & David, 160 (Lab.)
Currens, J. H. & Woodard, R. C., (339) (R.F.)
Curtis, A. C. & Owens, B. B., 486 (Der.)
Curtis, E. E., with Buckland, Dudgeon, Edward, Henderson-Begg, MacCallum, Niven, Rowlands, van den Ende, Bargmann & Shepherd, 436 (Typh.)
Cutting, C. C., with Hanzlik, 820 (Mal.)
Cutting, J. T., with Wright & Reppert, 781 (Heat Str.)
Czaczkes, J. W., with Olitzki, Oleinik & Kutzenok, (635) (Typh.)

D.

Daggy, R. H., 1107 (Mal.)
Dahms, R. G., with Olson, (798) (Ent.)
Daley, R., with Smart, 1160 (Sp.)
Damasceno, R. G., Causey, O. R. & Arouck, R., (1195) (Ent.)

Dame, L. R., 122 (Typh.)
Dammin, G. J., with Rogers, 1049 (Hel.)
——, with Weller, 347, 1155 (Hel.)
Damon, S. R., with Johnson, 915 (Typh.)
Danglade, J. H. & Fitzgerald, P. J., 576 (Hel.)
Daniel, L. J., with Robertson, Farmer, Norris & Heuser, (1066) (Haem.)
Daniels, W. B., with Mason, Paddock & Gordon, 1157 (Hel.)
Dansey-Browning, G. C. & Rich, W. M., 588 (Oph.)
Darby, W. J., 855 (Haem.)
—— & Jones, E., 949 (Sp.)
——, —— & Johnson, H. C., 667, 770 (Sp.)
Darius, D. J., 1182 (Oph.)
Das Gupta, B. M., Lowe, J. & Chakravarti, H., 298 (Mal.)
—— & Sen, R. L., 1145 (R.F.)
—— & Siddons, L. B., 4 (Mal.)
Das Gupta, C. R., Ganguli, S. & Chatterjea, J. B., 1066 (Haem.)
Dastidar, S. K. G., 318 (Leish.)
Datta, S., with Bose, Ghosh & Mitra, 1028 (Leish.)
Davalos, A., 381 (Misc. Dis.)
Davey, D. G., 712, 1010 (Mal.)
——, with Curd & Rose, 394, 400 (Mal.)
Davey, T. F., 1151 (Lep.)
Davey, T. H., with Adams, Maegraith, King, Townshend & Havard, 402 (Mal.)
——, with Maegraith, Adams, King, Townshend & Havard, 402 (Mal.)
David, J., (605) (Reports, etc.)
David, K., with Atchley, Bacon & Curran, 160 (Lab.)
David, W. A. L., 679, 960, 1192 (Ent.)
Davids, B., Jr., 124 (Typh.)
Davies, J. A., (939) (Hel.)
Davis, D. E. & Pollard, M., 1033 (Typh.)
Davis, D. H. S., 1038 (Pl.)
——, with de Meillon, 966 (Ent.)
Davis, D. J. & Sullivan, T. de S., 1026 (Tryp.)
Davis, G. E., with Mackie, Fuller, Knapp, Steinacker, Stager, Traub, Jellison, Millspaugh, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Davis, H. V. & Neff, F. C., 779 (Der.)
Davis, J. E., (952) (Haem.)
Davis, M. I., (822) (Mal.)
——, with Curd, Owen, Rose & Tuey, (1110) (Mal.)
Davis, O. T., Harrell, G. T. & King, E. S. (143) (Hel.)
Davis, W. A., 427 (Typh.)
Davison, A. R., 455 (Lep.)
Dawson, J., with Markson, 12 (Mal.)
Day, C. L., Wood, E. A. & Lane, W. F., (853) (Hel.)
Deane, L. M., with Causey & Deane, (199) (Mal.)
Deane, M. P., with —— & ——, (199) (Mal.)
Dearborn, E. H. & Marshall, E. K., Jr., 404 (Mal.)
Debray, J. R., with Hillemand, Varay & Duguet, 658 (Hel.)
Degazon, D. W., (62) (Haem.)
Degotte, J., 340 (Lep.)
Del Pozo, E. C., González Q., J. & Méndez, T. H., (148) (Vms.)
Del Valle Alemán, S., with Sotolonga & Alfonso, (50) (Hel.)
Delecourt, with Cavaillon, Bernard & Boyer, 544 (Typh.)
Delmanto, A., (602) (Ent.)
Delpy, L. P., (1080) (Ent.)
Delville, J., 95 (Mal.)
Demarest, C. R., with Heidelberger & Mayer, 816 (Mal.)
Denhoff, E., 145 (Haem.)
Dennell, R., (1195) (Ent.)
Dennison, W. & Evans, W., (959) (Misc. Dis.)
Deonier, C. C. & Burrell, R. W., 410 (Mal.)
——, ——, Maple, J. D. & Cochran, J. H., 101 (Mal.)
——, with Eide & Burrell, 600 (Ent.)
—— & Jones, H. A., 531 (Mal.)
——, with ——, Burrell & Knipling, 530 (Mal.)
——, with ——, Lindquist & Husman, (628) (Mal.)
——, Maple, J. D., Jones, H. A., Hinchey, E. & Eide, P. M., 101 (Mal.)
——, with Wisecup, 101 (Mal.)
——, with ——, Brothers & Eide, 1078 (Ent.)
——, with —— & Burrell, 530 (Mal.)
Derrick, E. H., with Morrissey, 31 (Typh.)
——, with Saxton & Hatcher, 1169 (Der.)
Deschiens, R., (352) (Hel.)
—— & Cottet, J., (766) (Hel.)
—— & Lamy, L., (352) (Hel.)
Deshmukh, M. D., 432 (Typh.)
Desportes, C., 759 (Hel.)
—— & Campana, Y., (746) (R.F.)
Despujols, B., Bergeret, C., Calmet, L. & Rouvier, J., (253) (Misc. Dis.)
Dethier, V. G., 598 (Ent.)
Deutschman, Z., 76 (Reports, etc.)
Devignat, R., 647, 1137 (Pl.)
——, Schoetter, M. & Gille-Simul, S., 330 (Pl.)
Dewar, H. A. & Walmsley, R., 136 (R.F.)
Dewey, H. M. & Wormall, A., 830 (Tryp.)
Dewhurst, K. E., 773 (Haem.)
Dharmendra, 452 (Lep.)
Dhayagude, R. G., 353 (Hel.)
Dias, E., 830 (Tryp.)
—— & Ferreira, L. B., (831) (Tryp.)
—— & de Freitas, L., Jr. 108 (Tryp.)
—— & Torrealba, J. F., 109 (Tryp.)
Diaz, I., 213 (Y.F.)
Díaz Atiles, A., 444 (Am.)
Díaz Rivera, R., with Hernández Morales, 230 (Hel.)
Diaz-Rivera, R. S., 925 (Den.)
——, with Suárez & Hernández Morales, 496 (Lab.)
——, with Pepper, 356 (Hel.)
Dick, G. W. A. & McCarthy, D. D., 660 (Hel.)
Dick, J. C., 726 (Typh.)
Dick, W. S., with Woofter & Bierring, 372 (Haem.)
Digo, R., with Mollaret, (653) (R.F.)
Dingledine, M. J., Willis, C. & Harrell, G. T., 837 (Typh.)
Dinh-Van-Thang, with Meyer-May, Ho-Dac-Di, Vu-Dinh-Tung & Tong-That-Tung, 87 (B.R.)
Discombe, G., 477 (Haem.)

Dixon, H. M., with Twining & Weidman, 674 (Der.)
Dixon, K. C., with Wood, 44 (R.F.)
Doan, C. A., with Wilson & Saslaw, (952) (Haem.)
Dobes, W. L. & Jones, J., 672 (Der.)
Doeleman, H., 875 (B.R.)
Doerr, W., 123 (Typh.)
Dolce, F. A. & Franklin, J. E., 230 (Hel.)
Dole, V. P. & Emerson, K., Jr., with Braun, E., 624 (Mal.)
Donatien, A., with Sergent & Parrot, (603) (Ent.)
Donegan, E. A., 550 (Typh.)
Donovick, R., Farrell, M. & Smith, F., 635 (Typh.)
——, with Groupé, 635, 726 (Typh.)
Dorin, R. P., 1057 (Hel.)
Dormer, B. A., 665 (Def. Dis.), 843 (Am.)
Dostrovsky, A., 320 (Leish.)
—— & Sagher, F., 1030 (Leish.)
Douglas, J. R., with Wheeler, 35 (Pl.)
Doull, J. A., Guinto, R. C., Rodriguez, J. N. & Bancroft, H., 453 (Lep.)
Dow, D., 460 (Lep.)
Dow, R. S., 678 (Prot.)
Dowling, P. G., 786 (Misc. Dis.)
Downs, W. G., 1004 (Mal.)
—— & Pittendrigh, C. S., 1001 (Mal.)
Drew, W. R. M., 684 (Reports, etc.)
Drewe, F., 498 (Reports, etc.)
Drouet, P. L., Thomas, C., Herbeuval, R. & Faivre, G., 467 (Hel.)
Druckmann, A. & Schorr, S., 133 (Am.)
Drysdale, G. F., with Brodie, Cook & McIntosh, 841 (Dys.)
Duane, R. B., with Rose & Fischel, 433 (Typh.)
Dubois, A., 1087 (B.R.)
Ducharme, P., with Golden, 651 (Am.)
Dudgeon, A., with Buckland, Edward, Henderson-Begg, MacCallum, Niven, Rowlands, van den Ende, Bargmann, Curtis & Shepherd, 436 (Typh.)
Dueñas, C. & Abad, R., 935 (Lep.)
Duensing, F., 211 (Typh.)
Duff, F. L., with Howe, 523 (Mal.)
Duguet, with Hillemand, Varay & Debray, 658 (Hel.)
Dukhanina, N. N., 999 (Mal.)
Dulaney, A. D., with Babin, 297 (Mal.)
—— & Watson, R. B., 818 (Mal.)
Duncan, G. G., 708 (Mal.)
Dunlop, E. E., 558 (Am.)
Dunn, R. C., 1081 (Lab.)
Duren, A., 954 (Vms.)
Durieux, C., 190 (Rab.)
Dutta, D. K., with Clark, 559 (Am.)
Dutta, N. K., Mukerji, B. & Sekar, C. C., 1029 (Leish.)
Duvalier, F., 448 (Ys.)
Duvolon. S. Mlle., with Roubaud & Stefanopoulo, 419 (Tryp.)
——, with Stefanopoulo & Caubet, 417, 420 (Tryp.)
Dwindelle, J. H., Rein, C. R., Sternberg, T. H. & Sheldon, A. J., 1043 (Ys.)
van Dyke, H. B., with Gellhorn, Pyles & Tupikova, 722 (Leish.)

E.

Eads, R. B., 1108 (Mal.)
Eagle, H., 421, 1017 (Tryp.)
——, with Schatz, Magnuson & Waksman, 1025.
Eason, J. L., Jr., with Johnson, 1111 (Mal.)
Ebeling, W., 680 (Ent.)
Ecke, R. S., Gilliam, A. G., Snyder, J. C., Yeomans, A., Zarafonetis, C. J. & Murray, E. S., 545 (Typh.)
——, with Yeomans, Snyder, Murray & Zarafonetis, 636 (Typh.)
——, with Zarafonetis, Yeomans, Murray & Snyder, 912 (Typh.)
Ecke, W., 116 (Typh.)
Eckstein, A. & Nixon, W. C. W., 623 (Mal.)
Edington, A. D., 42 (Am.)
Edison, A. O., Silber, R. H. & Tennent, D. M., 363 (Def. Dis.)
Edny, E. B., 966 (Ent.)
Edward, D. G. ff, with Buckland, Dudgeon, Henderson-Begg, MacCallum, Niven, Rowlands & Van den Ende, Bargmann, Curtis & Shepherd, 436 (Typh.)
Edwards, C., 659 (Hel.)
Egrejas, G., with Ferreira, 442 (Am.)
Ehrman, F. C., with Young, Stubbs, Moore, Hardman, Ellis & Burgess, 5 (Mal.)
Eichhorn, F., with Rappaport, (1164) (Haem.)
Eichler, W., (96) (Mal.)
Eide, P. M., Deonier, C. C. & Burrell, R. W., 600 (Ent.)
——, with ——, Maple, Jones & Hinchey, 101 (Mal.)
—— with Wisecup & Brothers, 864 (Ent.)
——, with ——, —— & Deonier, 1078 (Ent.)
Einhorn N. H. with Whittier & Miller, 577 (Hel.)
Eklund, C., with Adams & Horns, 862 (Prot.)
Elder, J. H., 498 (Reports, etc.)
Eliot, T. S., with Knisely & Bloch, 631 (Mal.)
——, with ——, Stratman-Thomas & Bloch, 1113 (Mal.)
Elkes, J. T., with Cooke, Frazer, Parkes & Peeney, 946 (Sp.)
El Kordy, with Halawani, (1143) (Am.)
——, with Latif, (1158) (Hel.)
Ellerbrook, L. D., Lippincott, S. W., Cateno, C. F., Gordon, H. H. & Marble, A., 626 (Mal.)
——, with ——, Hesselbrock, Carrico & Marble, 1009 (Mal.)
——, with ——, ——, Gordon, Gottlieb & Marble, 523 (Mal.)
Ellis, J. M., with Young, Stubbs, Moore, Ehrman, Hardman & Burgess, 5 (Mal.)
Elsaesser, K.-H., 228 (Hel.)
Elson, K. A., Pepper, D. S. & Forrester, J. S., 556 (Dys.)
El-Tobgy, A. F., (1184) (Oph.)
Elvehjem, C. A., with Cooperman, McCall & Ruegamer, 772 (Haem.)
——, with —— & Ruegamer, (1067) (Haem.)
Emerson, K., Jr., with Dole & Braun, 624 (Mal.)
Emert, J. T., with Cohen & Goss, 595 (Misc. Dis.)

Engel, R., 111 (Tryp.)
Engledow, F. L., 72 (Reports, etc.)
——, with Moyne, Stubbs, Crowdy, Citrine, Mackinnon, Blacklock & Henderson, 70 (Reports, etc.)
English, J. C., 516 (Mal.)
English, J. P., Clark, J. H., Shepherd, R. G., Marson, H. W., Krapcho, J. & Roblin, R. O., Jr., (905) (Mal.)
English, R. B., 220 (Am.), 243 (Haem.)
Engstrom, W. W., with Gordon, Marble, Brunsting & Lippincott, 1007 (Mal.)
Epidemiological Information Bull. (UNRRA Health Division.) Wash., 124 (Y.F.)
Epstein, E., 479 (Der.)
Errecart, L. M., 1071 (Der.)
Evans, E. A., Jr., with Moulder, 1118 (Mal.)
——, with Speck & Moulder, 1116 (Mal.)
Evans, E. R., with Agress, 549 (Typh.)
Evans, R. L., with Smith, Stormont & Bianco, (1157) (Hel.)
Evans, R. W., 371 (Haem.)
Evans, W., with Dennison, (959) (Misc. Dis.)
Ewing, F. M., with Haas & Wilcox, 1114 (Mal.)
Exemplarskaya, E. V., 1113 (Mal.)
Eyles, D. E., Sabrosky, C. W. & Russell, J. C., 199 (Mal.)

F.

Faget, G. H., (453) (Lep.)
—— & Pogge, R. C., 343, 460 (Lep.)
Fairchild, G. B. & Barreda, E. A., 680 (Ent.)
Fairchild, L. M., 329 (Den.)
Fairley, N. H., 527, 529, 628 (Mal.), 1132 (Typh.)
Faivre, G., with Drouet, Thomas & Herbuval, 467 (Hel.)
Fales, J. H., with McGovran & Goodhue, 962 (Ent.)
Falin, L. I., 24 (Typh.)
Familiari, P., (1184) (Oph.)
Farber, B., (1067) (Haem.)
Farinaud, E. & Prost, P., (285) (Mal.)
Farinaud, M. E., 247 (Heat Str.)
Faris, R. A., (263) (Misc. Dis.)
Farmer, F. A., with Robertson, Daniel, Farmer & Norris, (1066) (Haem.)
Farrell, M., with Donovick & Smith, 635 (Typh.)
Faust, E. C., 286 (Mal.), 758 (Hel.), 960 (Ent.)
——, with Craig, 506 (B.R.)
——, Wright, W. H., McMullen, D. B. & Hunter, G. W., 756 (Hel.)
Favarel, R., 329 (Y.F.)
Favour, C. B., with Weber, 339 (R.F.)
Fay, F. R. & Susman, E., 781 (Heat Str.)
Fay, R. W., Simmons, S. W. & Clapp, J. M., 790 (Ent.)
Federated Malay States : Inst. for Med. Res., 1106 (Mal.)
Fedotov, P. I., 12 (Mal.)
Fehily, L., (61) (Def. Dis.)
Feigenbaum, A. & Kornblueth, W., 1074 (Misc. (Dis.)
Felix, A., 1132 (Typh.)
Felsen, J., 1089 (B.R.)
Felsenfeld, O. & Young, V. M., 332 (Chl.)
Felton, H. L., Barnes, R. C. & Wilson, A., 308 (Mal.)
Feng, Y. S., with Pang, Zia & Chen, 326 (Typh.)
Fennah, R. G., 865 (Ent.)
Fennel, E. A., 458 (Lep.)
Fenton, J. C. B. & Innes, J., 298 (Mal.)
Ferguson, F. F., with Arnold & Upholt, 793 (Ent.)
Ferguson, M. S., with Bang, Hairston & Graham, 350 (Hel.)
Fergusson, A. G., 1165 (Der.)
Fernandez, J. C., with Niño, (257) (Ent.)
Fernández, J. M. M. & Soto, C. M., 850 (Lep.)
Fernando, A. S., Ayuyao, C. D. & Cruz., J. N., 1181 (Oph.)
Ferreira, F. da C. & Egrejas, G., 442 (Am.)
Ferreira, J. A., with Alecha & Carpanelli, (136) (Am.)
Ferreira, L. B., with Dias, (831) (Tryp.)
Ferrero, A., (329) (Y.F.)
Fidler, H. K., with Oastler, 1029 (Leish.)
Fielding J. W. 1147, 1154 (Lep.)
Fiennes, R. N. T. W., 16 (Tryp.)
Filatov, V. P., 724 (Leish.)
Findlay, G. M., 485 (Der.)
Finlayson, M. H. & Hollow, K., 376 (Vms.)
Finnegan, S., 262 (B.R.)
Fiol, H., 848 (Lep.)
Fischel, E. E., with Rose & Duane, 433 (Typh.)
Fischer, F. K., 465 (Hel.)
Fischer, L., 259 (Reports, etc.)
Fischer, R., with Boventer, 546 (Typh.)
Fishbon, H. M., 1046 (Hel.)
Fisher, A. C. & Lendrum, A. C., 1186 (Misc. Dis.)
Fisk, F. W., with Knowles, 529 (Mal.)
Fite, G. L. & Gemar, F., 751 (Lep.)
Fitzgerald, P. J., with Danglade, 576 (Hel.)
Fitzhugh, O. G., Nelson, A. A. & Calvery, H. O., with Glassman, J. M., 525 (Mal.)
Fitzpatrick, F. K., 540 (Typh.)
Flanagan, H. F., with Iams & Tenen, 587 (Der.)
Fletcher, F., with Wilkinson & Israels, 1065 (Haem.)
Floch, H., 820 (Mal.)
—— & Abonnenc, E., 702, 703 (Mal.)
—— & de Lajudie, P., 342, 933 (Lep.), 352, (852), 1155 (Hel.), 704 (Mal.), 908 (Tryp.)
Florio, L., Hammon, W. McD., Laurent, A. & Stewart, M. O., 737 (Den.)
——, Stewart, M. O. & Mugrage, E. R., 438 (Den.)
Fonseca, J. A. B. & Unti, O., (905) (Mal.)
Fontes, A., with Puig Solanes & Antonio Quiroz, 764 (Hel.)
Fonzari, M., 62 (Der.)
Forbes, F. A., with Hobbs, 1179 (Oph.)
Foreign Office, 34 *bis* (Y. F.)
Forrester, J. S., with Elson & Pepper, 556 Dys.)
Fourman, L. P. R., with Black & Trinder, 667 (Sp.)
Fourman, P., with Black, 474 (Sp.)
——, with —— & Trinder, 1161 (Sp.)
Fowler, E. B., with Avera, Yow & Farrell, 765 (Hel.)

Fowweather, F. S. & Anderson, W. N., 1064 (Sp.)
Frachtman, H. J. & McCollum, W. T., 493 (Misc.)
Franchel, F., with Benda, 1198 (Lab.)
Frandsen, S. & Kringelbach, J., 1163 (Sp.)
Franke, M., with Ciuca, Mesrobeanu, Badenski & Munteanu, (210) (Typh.)
Franklin, J. E., with Dolce, 230 (Hel.)
Franklin, K. J. *et al.*, 905 (Mal.)
Franz, K., with Weinman, 207 (Tryp.)
Fraser, P., 75 *bis* (Reports, etc.)
Fraser-Shearer, W., 498 (Reports, etc.)
Frazer, A. C., (1161) (Sp.)
——, with Cooke, Elkes, Parkes & Peeney, 946 (Sp.)
Fredericks, M. G. & Hoffbauer, F. W., 97 (Mal.)
Freedberg, A. S. & Riseman, J. E. F., 245 (Vms.)
Freedman, B. J., 855 (Haem.)
Freeman, G., 839 (Y.F.)
Freeman, R. B., 682 (Ent.)
Freire, S. A., with Magalhães, (1123) (Tryp.)
de Freitas, J. L. P., (910) (Tryp.)
de Freitas, L., Jr., with Dias, 108 (Tryp.)
French, M. H., with Wilde, 1120 (Tryp.)
Freund, J., Sommer, H. E. & Walter, A. W., 311 (Mal.)
——, Thomson, K. J., Sommer, H. E., Walter, A. W. & Schenkein, E. L., 311 (Mal.)
Friedheim, E. A. H. & Berman, R. L., 1124 (Tryp.)
Friedlander, R. D., with Sweeney & Queen, 114 (Leish.)
Fritz, R. F., with Bradley, 198 (Mal.)
Frommeyer, W. B., with Spies, Garcia Lopez, Lopez Toca & Gwinner, 949 (Sp.)
——, with ——, Perry & Cogswell, 60 (Def. Dis.)
——, with ——, Vilter & Cline, 772 (Haem.)
Fuller, H. S., with Mackie, Davis, Knapp, Steinacker, Stager, Traub, Jellison, Millspaugh, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Fulton, F. & Joyner, L., 435 (Typh.)
Fulton, J. D. & Goodwin, T. W., 17 *bis* (Leish.)
—— & Harrison, C. V., 1123 (Tryp.)
—— & Stevens, T. S., 1121 (Tryp.)
Funke, A., Bovet, D. & Montézin, G., 713 (Mal.)
——, with Lwoff & Bovet, 313 (Tryp.)

G

Gaase, A., 236 (Hel.)
Gahan, J. B. & Lindquist, A. W., 99 (Mal.)
——, Travis, B. V. & Lindquist, A. W., 99 (Mal.)
——, ——, Morton, F. A. & Lindquist, A. W., 99 (Mal.)
Gaines, T. B., with Upholt, Simmons & Arnold, 794 (Ent.)
Gale, G. W., 1083 (Reports, etc.)
Gallais, P., 633 (Tryp.)
Gallardo, F. P., with Clavero, (117) (Typh.)
Gallut, J., with Grabar, 129 (Chl.)
Galushkina, O. N., 300 (Mal.)
Gama, C. & de Sá, J. M., 936 (Hel.)
Gamal Nor El Deen, with Khalil Bey & Halawani, 50 (Hel.)
Ganguli, S., with Das Gupta & Chatterjea, 1066 (Haem.)
Garcia Lopez, G., with Spies, Frommeyer, Lopez Toca & Gwinner, 949 (Sp.)
Garcia Sastre, L., 283 (Mal.)
Garland, H. G., 588 (Oph.)
Garland, L. H., with Sokolow, 432 (Typh.)
Garnham, P. C. C., 291 (Mal.)
——, Harper, J. O. & Highton, R. B., 736 (Y.F.)
Gärtner, W., 211 (Typh.)
Gartrell, F. E., with Kruse, (308) (Mal.)
Garzón, R. & Argüello Pitt, L., 849 (Lep.)
Gaschen, H., 160 (Ent.), 313 *ter* (Tryp.)
Gass, H., with Weyrauch, 737 (Den.)
Gast G., A. & Bates, M., 644 (Y.F.)
Gast-Galvis, A., 924 (Y.F.)
—— & Rengifo, S., 113 (Leish.)
Gaumond, E., with Berger & Beadry, 150 (Der.)
Gavin, L D. M., (1015) (Mal.)
Gear, J., 632 (Bl.)
Geiger, J., with Sharnoff & Selzer, 1002 (Mal.)
Geiling, E. M. K., with Chen, 315 (Tryp.)
——, with Kelsey & Oldham, 404 (Mal.)
——, with ——, —— & Cantrell, (625) (Mal.)
Gelfand, M., (200) (Mal.), 1049 (Hel.)
Gellhorn, A., van Dyke, H. B., Pyles, W. J. & Tupikova, N. A., 722 (Leish.)
Gemar, F., with Fite, 751 (Lep.)
Getz., L., 57 (Hel.)
Gevorkov, A. A., 720 (Leish.)
Gezon, H. M., with Greaves & Alston, 445 (R.F.)
Ghose, N., with Sen, 576 (Hel.)
Ghose, R. N., with Gupta, Chatterjee & Paul, 130 (Chl.)
Ghosh, B. K., with Majumder & Bagchi, 441 (Dys.)
Ghosh, B. N., 504 (B.R.)
Ghosh, H., Ghosh, N. N. & Ray, J. C., 1125 (Leish.)
Ghosh, J. K., with Bose, 36 (Dys.)
——, with —— & Rakshit, 928 (Dys.)
Ghosh, L. M. & Panja, D., (490) (Misc.)
Ghosh, N. N., with Ghosh & Ray, 1125 (Leish.)
Ghosh, S. K., with Panja, 218 (Chl.)
Ghosh, T. N., with Bose, Mitra & Datta, 1028 (Leish.)
Ghul, R., (283) (Mal.)
Gibson, Q. H. & Harrison, D. C., 773 (Haem.)
Giglio, C., (710) (Mal.)
Giglioli, G., 286 (Mal.)
Gilchrist, B. M., with Bishop, 710 (Mal.)
Gilchrist, M., with King & Tarnoky, (707) (Mal.)
——, with —— & Wootton, 822 (Mal.)
Gilje, L. E. & Lampson, R. S., (444) (Am.)
Gille-Simul, S., with Devignat & Schoetter, 330 (Pl.)
Gillette, H. P. S., 824 (Mal.)
Gillham, E. M., with Potter, 1193 (Ent.)

Gilliam, A. G., with Ecke, Snyder, Yeomans, Zarafonetis & Murray, 545 (Typh.)
——, with Yeomans & Snyder, 24 (Typh.)
Gillman, J. & Gillman, T., 364 (Def. Dis.)
——, with ——, 59, 237 (Def. Dis.)
——, —— & Brenner, S. 365, 366 (Def. Dis.)
——, Mandelstam, J. & Gillman, T., 666 (Def. Dis.)
Gillman, T. & Gillman, J., 59, 237 (Def. Dis.)
——, with —— 364 (Def. Dis.)
——, with —— & Mandelstam, 666 (Def. Dis.)
——, with —— & Brenner, 365, 366 (Def. Dis.)
Ginsberg, J. E. & Shallenberger, P. L., 1007 (Mal.)
Girão J., with de Meira & Coito, (760) (Hel.)
Girard, G., 439 (Pl.)
Girard, J., with Simonin, (478) (Haem.)
Giroud, M.-L., with Giroud, 322 (Typh.)
Giroud, P., 913 (Typh.)
——, with Bablet, (322) (Typh.)
—— & Giroud, M.-L., 322 (Typh.)
—— & Sureau, B., (324) (Typh.)
Girsham, J. A. V., with Mackie, Davis, Fuller, Knapp, Steinacker, Stager, Traub, Jellison, Millspaugh, Austrian, Bell, Kohls & Wei Hsi, 917 (Typh.)
Giua, A., (623) (Mal.)
Glasgow, J. P., with Melville, Wilson & Hocking, 92 (Mal.)
Glasgow, R. D. & Collins, D. L., 924, (Typh.), 962 (Ent.)
Glass, F. A., with Weidman, 955 (Der.)
Glassman, J. M., with Fitzhugh, Nelson & Calvery, 525 (Mal.)
Gleeson-White, M. H., with Hutchison & Pippard, 338 (R.F.)
Gluck, A. C., with Kaplan, 438 (Y.F.)
Gold, M. A., with Yuile & Hinds, 531 (Bl.)
Goldberg, L. C., 776 (Der.)
Goldeck, H. & Walther, R., 215 (Den.)
Golden, A. & Padilla, E., 1185 (Ulc.)
Golden, F., with Moore, Kessel, Simonsen, Marmorston, Llewellyn, Kaplan, Anderson & Jaffe, (650) (Dys.)
Golden, R. & Ducharme, P., 651 (Am.)
Golding, F. D., 965 (Ent.)
Goldman, L. & Ortiz, L. F., 664 (Hel.)
Goldstein, B., (1077) (Misc. Dis.)
Gomez, J. E., 956 (Der.)
Gomez Lucas, R., with Guillen Prats, 936 (Lep.)
González, Q., J., with Del Pozo & Méndez, (148) (Vms.)
González, R. D., 163 (Reports, etc.)
González Prendes, M. A., with Ibarra Pérez, 561 (Ys.), 226, 563, 933 (Lep.)
——, with Trespalacios, 934 (Lep.)
González Rincones, P., 937 (Hel.)
Goodale, R. H., with Allen, 856 (Der.)
Goodhue, L. D., with McGovran & Fales, 962 (Ent.)
—— & Riley, R. L., 962 (Ent.)
Goodman, A. A., Weinberger, E. M., Lippincott, S. W., Marble, A. & Wright, W. H., 573 (Hel.)
Goodwin, L. G., 318, 319 (Leish.)
—— & Marshall, P. B., 315 (Tryp.)
Goodwin, T. W., with Fulton, 17 *bis* (Leish.)
Gopalan, C. 768, 1160 (Def. Dis.)
Gordon, H. H., with Ellerbrook, Lippincott, Cateno & Marble, 626 (Mal.)
——, with Lippincott, Ellerbrook, Hesselbrock, Gottlieb & Marble, 523 (Mal.)
—— Marble, A., Engstrom W. W., Brunsting, H. A. & Lippincott, S. W., 1007 (Mal.)
——, with Mason, Daniels & Paddock, 1157 (Hel.)
Gordon, R. M. 684 (Reports, etc.)
——, with Bertram & Unsworth, 1197 (Ent.)
——, with Unsworth, 1195 (Ent.)
Goren, M. L., 779 (Der.)
Goss, C. C., with Cohen & Emert, 595 (Misc. Dis.)
Gottfried, S. P. & Levine, A. C., 301 (Mal.)
Gottlieb, L. with Lippincott, Ellerbrook, Hesselbrock, Gordon & Marble, 523 (Mal.)
Gouck, H. K., with Smith, (494), 1198 (Ent.)
Gould, S., 882 (B.R.)
Gould, S. E., 362, 578 (Hel.)
Goyal, R., with Mathur, 217 (Pl.)
Grabar, P. & Gallut, J., 129 (Chl.)
Graham, O. H., with Bang, Ferguson & Hairston, 350 (Hel.)
Graña, A., 139 (Hel.)
Graña, R., with Rocha e Silva, 1052 *bis* (Hel.)
Granett, P. & Haynes, H. L., 681 (Ent.)
Grant, W. M., 1176 (Oph.)
Grasset, E., 1068 (Vms.)
——, Schaafsma, A. & Hodgson, J. A., 670 1069 (Vms.)
Grau Triana, J., (225) (Lep.)
Greaves, D. P., with Smith, 965 (Ent.)
Greaves, F. C., Gezon, H. M. & Alston, W. F., 445 (R.F.)
Green, R. A., with Webb, 598 (Ent.)
Greene, C. C., Jr., with Noe & Cheney, 521 (Mal.)
Greenberg, M., with Birnkrant & Most, 221 (Am.)
Greenberg, S. with Sulzberger, Addenbrooke, Joyce & Mack, 1166 (Der.)
Greiff, D. & Pinketon, H., 22 (Typh.)
Greig, E. D. W., (380) (Misc. Dis.), 927 (Chl.)
Grenoilleau, G., 22 (Typh.), 929 (R.F.)
Grier G. S. with Borden & Waddill 151 (Heat Str.)
Griffiths, J. T., Jr., 431 (Typh.)
Grocott, R. G., with Kean, 158 (Prot.)
Groupé, V. & Donovick, R., 635, 726 (Typh.)
Grübel, W., 118 (Typh.)
Gruenfelder, B. & Lasch, W., 1148 (Lep.)
Guadagnini, M., 851 (Lep.)
Guha, G. S., (1109) (Mal.)
——, with Bhattacharjya, 1178 (Oph.)
Guichard, F., 343 (Lep.)
Guilhon, J., (356) (Hel.)
Guillen Prats, J. & Gomez Lucas, R., 936 (Lep.)
Guimarães, F. N., 46, 451, 1145 (Ys.)
——, with da Cunha, 562 (Ys.)
Guinto, R. S., with Doull, Rodriguez & Bancroft, 453 (Lep.)
——, with Bancroft, Rodriguez & Marques, 454 (Lep.)

Gunn, D. L., Kirk, R. L. & Waterhouse, J. A. H., 502 (Reports, etc.)
Gupta, J. C., Panja, G. & Chatterjee, M., 1138 (Pl.)
Gupta, S. K., Chatterjee, B. C., Paul, B. M. & Ghose, R. N., 130 (Chl.)
Gutiérrez Sánchez, E., 1110 (Mal.)
Gwinner, G., with Spies, Frommeyer, Garcia Lopez & Lopez Toca, 949 (Sp.)

H.

Haas, V. H., Wilcox, A. & Ewing, F. M., 1114 (Mal.)
Habel, K., 192 (Rab.)
Habibi, M., 1129 (Typh.)
Hackett, C. J., 835 (Typh.)
Haddad, N. N., 379 (Misc. Dis.)
Haddow, A. J., 79, 80 (Reports, etc.), 255, 863 (Ent.)
Haig, D. A., with Polson & Joubert, (954) (Vms.)
Hailey, H., 954 (Der.)
Hairston, N. G., with Bang, Ferguson & Graham, 350 (Hel.)
——, with Trager & Bang, 821 (Mal.)
Halawani, A. & El Kordy, M. I., (1143) (Am.)
——, with Khalil Bey & Gamal Nor El Deen, 50 (Hel.)
—— & Nor-el-Din, G., 1109 (Mal.), (1145) (Ys.), (1158) (Hel.)
——, —— & Awni, A. Y., 1142 (Am.)
Halawany, A. & Jalili, M., 540 (Leish.)
Hall, B. & Billings, J. J., 443 (Am.)
Hamilton, H. L., 320, 434, 734 (Typh.)
Hamilton, T. S., with Johnson & Mitchell, 591 (Heat Str.)
——, with Shields, Johnson & Mitchell, 591 (Heat Str.)
——, with Spector, (378) (Heat Str.)
——, with —— & Mitchell, (378), 591 *bis.* (Heat Str.)
Hamilton Paterson, J. L., with Piney, 870 (B.R.)
Hammon, W. McD., with Florio, Laurent & Stewart, 737 (Den.)
Hampton, B. C., 554 *bis* (Pl.)
Hamzah, N. A., 651 (Am.)
Hanzlik, P. J. & Cutting, C. C., 820 (Mal.)
Hardcastle, A. B., with Wharton, 1080 (Ent.)
Harding, R. D., 105 (Tryp.)
Hardman, N. F., with Young, Stubbs, Moore, Ehrman, Ellis & Burgess, 5 (Mal.)
Hardman, N. H., with Moore, Young & Stubbs, 1002 (Mal.)
Hargeaves, W. H., 844, 1040 (Am.)
——, with van den Ende, Locket, Niven & Lennhoff, 918 (Typh.)
Harley R. D. 1148 (Lep.)
Harper, F., 510 (B.R.)
Harper, J. O., with Garnham & Highton, 736 (Y.F.)
Harrell, G. T., with Avera, Yow & Fowler, 765 (Hel.)
——, with Davis & King, (143) (Hel.)
——, with Dingledine & Willis, 837 (Typh.)
—— & Horne, S. F., 655 (Lep.)
——, Wolff, W. A., Venning, W. L. & Reinhart, J. B., 923 (Typh.)
Harris, J. R. & Hickey, M. D., 138 (Hel.)
Harris, J. S. & Summers, W. A., 569 (Hel.)
Harrison, C. V., with Fulton, 1123 (Tryp.)
Harrison, D. C., with Gibson, 773 (Haem.)
Harrison, G. F., 945 (Def. Dis.)
Harvey, A. M., Kuffler, S. W. & Tredway, J. B., 490 (Misc. Dis.)
Haskins, W. T., with Lawton, Brady & Ness, 55 (Hel.)
Hasseltine, H. E., (453), 457 (Lep.)
Hatcher, F., with Saxton & Derrick, 1169 (Der.)
Hatoff, A., 682 (Ent.)
Hauser, C. R., with Breslow, Walker, Yost & Shivers, (406) (Mal.)
Havard, R. E., with Adams, Maegraith, King, Townshend & Davey, 402 (Mal.)
——, with Maegraith, Adams, King & Millet, 238 (Sp.)
——, with ——, ——, ——, Townshend & Davey, 402 (Mal.)
Haveaux, G., 1019 (Tryp.)
Haviland, J. W., with Spector & Coggeshall, 823 (Mal.)
Hawe, P., 134, 1140 (Am.)
Hawking, F., 380 (Misc. Dis.), 410 (Mal.)
—— & Burroughs, A. M., (940) (Hel.)
Hay, S., 384 (Lab.)
Hayes, W., with Coxon, 406 (Mal.)
Hayman, J. M., Jr., with Behm, 762 (Hel.)
——, with Most, 1004, 1005 (Mal.)
——, with ——, London, Kane, Lavietes & Schroeder, 1011 (Mal.)
Haynes, H. L., with Granett, 681 (Ent.)
Hayward, G. W., with Cottrell, 653 (Am.)
Hazelton, A. R., 1181 (Oph.)
Headley, N. E., with Boland & Hench, 597 (Misc. Dis.)
Heath, P. & Zuelzer, W. W., 67 (Prot.)
Heaton, C. E., (839) (Y.F.)
Hegsted, D. M., with Williamson, McKibbin & Stare, 1009 (Mal.)
Heidelberger, M., Mayer, M., Alving, A. S., Craige, B., Jr., Jones, R., Jr., Pullman, T. N. & Whorton, M., 1013 (Mal.)
——, Coates, W. A. & Mayer, M. M., 1013 (Mal.)
——, Mayer, M. M. & Demarest, C. R., 816 (Mal.)
——, Prout, C., Hindle, J. A. & Rose, A. S., 1013 (Mal.)
Heilig, R., 520 (Mal.), 1074 (Misc. Dis.)
Hellerman, L., with Bovarnick & Lindsay, 824, 825 (Mal.)
Hellier, F. F., 835 (Typh.)
Hemmings, R. J., (1015) (Mal.)
Hemphill, E. C., with Schuhardt, 748 (R.F.)
Hench, P. S., with Boland & Headley, 597 (Misc. Dis.)
Henderson, D. G., with Rifkin, Cekada, Zarrow & Whitehead, 188 (Rab.)
Henderson, H. D., with Moyne, Stubbs, Crowdy, Citrine, Mackinnon, Blacklock & Engledow, 70 (Reports, etc.)
Henderson, H. J., 457 (Lep.)
Henderson, J. M., (205), 287 (Mal.)
Henderson, R. G., with Bengston & Topping, 19 (Typh.)

Henderson, R. G. & Topping, N. H., 20 (Typh.)
——, with —— & Bengston, 20, 21 (Typh.)
Henderson-Begg, A., with Buckland, Dudgeon, Edward, MacCallum, Niven, Rowlands & van den Ende, Bargmann, Curtis & Shepherd, 436 (Typh.)
Henrard, C. with van Hoof, Lewillon, Peel & Rodjestvensky, 536 (Tryp.)
——, with Wanson, 232 (Hel.)
——, with —— & Peel, 233 (Hel.)
Herbeuval, R., with Drouet, Thomas & Faivre, 467 (Hel.)
Herbut, P. A. & Kinsey, F. R., 959 (Misc. Dis.)
Hermans, E. H., 799 (Reports, etc.)
Hermitte, L. C. D., 970 (Reports, etc.)
Hernández Morales, F., (228), 462, (1158) (Hel.), 239 (Sp.)
—— & Diaz Rivera, R., 230 (Hel.)
—— & Oliver González, J., 1156 (Hel.)
——, —— & Pratt, C. K., 1045 (Hel.)
—— & Ruiz Cestero, G., (228) (Hel.)
——, with Suárez, 349 (Hel.)
——, with —— & Díaz Rivera, 496 (Lab.)
Hersh, J., (1190) (Misc. Dis.)
Hess, A. D., with Metcalf, Smith, Jeffery & Ludwig, 102 (Mal.)
Hesselbrock, W. B., with Lippincott Ellerbrook, Carrico & Marble, 1009 (Mal.)
——, with ——, Gordon, Gottlieb & Marble, 523 (Mal.)
Heuser, G. F., with Robertson, Daniel, Farmer & Norris, (1066) (Haem.)
Hickey, M. D., with Harris, 138 (Hel.)
Hicks, J. D., 1134 (Typh.)
Higgins, E., with Cottam, 1014 (Mal.)
Highton, R. B., with Garnham & Harper, 736 (Y.F.)
Hill K. R., 835 (Typh.)
Hill, R. B. & Hill, C. M., 1191 (Ent.)
Hillemand, P., Varay, A., Debray, J. R. & Duguet, Mme., 658 (Hel.)
Hilmy, I. S., 51 (Hel.)
Hinchey, E., with Deonier, Maple, Jones & Eide, 101 (Mal.)
Hindle, J. A., with Heidelberger, Prout & Rose, 1013 (Mal.)
Hinds, E. G., with Yuile & Gold, 531 (Bl.)
Hitti, J. K. & Khairallah, A. A., 1137 (Den.)
Hoare, C. A., 317 (Leish.)
Hobbs, H. E. & Forbes, F. A., 1179 (Oph.)
Hocking, K. S., 681 (Ent.)
——, with Melville, Wilson & Glasgow, 92 (Mal.)
Ho-Dac-Di, with Meyer-May, Vu-Dinh-Tung, Tong-That-Tung & Dinh-Van-Thang, 87 (B.R.)
Hodes, P. J. & Wood, F. C., 66 (Misc. Dis.)
Hodge, E. H. V., 48 (Hel.), 684 (Reports, etc.)
Hodgson, J. A., with Grasset & Schaafsma, 670 (Vms.)
Hoehn, D., 256 (Ent.)
Hoffbauer F. W., with Cook, 819 (Mal.)
——, with Fredericks, 97 (Mal.)
Hoffman, A. M., with Butt, 63 (Der.)
Hoffman, W. A., with Krakower & Axtmayer, 227 (Hel.)
Hollender, A. J., with Parker, 186 (Rab.)
Hollins, C., 1132 (Typh.)
Hollow, K., with Finlayson, 376 (Vms.)
Holloway, G. D., with Bent, Turner & Cuff, 253 (Misc. Dis.)
Holmes, E. G., 146 (Haem.)
Honig, P. & Verdoorn, F., 884 (B.R.)
van Hoof, L., (969) (Reports, etc.)
——, Lewillon, R., Henrard, C., Peel, E. & Rodjestvensky, B., 536 (Tryp.)
Horack, H. M., with Simmons, Whayne, Anderson & collaborators, 86 (B.R.)
Horecker, B. L., 1065 (Haem.)
Horgan, E. S. & Satti, M. H., 17 (Leish.)
Horne, S. F., with Harrell, 655 (Lep.)
Hornibrook, J. W., 741 (Pl.)
Horns, R., with Adams & Eklund, 862 (Prot.)
Hornsby, S., with Clemo & Cocker, (1054) (Hel.)
Horvath, S. M., with Shelley, 675 (Heat Str.)
Hoskins, W. M., with Magy, 601 (Ent.)
Howe, C. D. & Duff, F. L., 523 (Mal.)
Howell, K. M., with Knoll, 444 (Am.)
Howell, W. L., 327 (Typh.)
Howells, G., (334) (Am.)
Howser, J. W., with Vaughn & Shropshear, (157) (Misc. Dis.)
Hoyem, H. M., with Bozicevich & Walston, 798 (Lab.)
Hubbard, C. S., 498 (Reports, etc.)
Hudson, E. H., 931 (Ys.)
Huff, C. G. & Coulston, F., 1115 (Mal.)
——, with —— & Cantrell, 205 (Mal.)
Huffaker, C. B., with Rey & Soto, (816) (Mal.)
——, Soto, H., & Rey, H., 392 (Mal.)
Hughes, C. O., with Brackett, 15 (Mal.)
Hughes, W., 581, 766, 767, 1062 (Def. Dis.)
Hühne, W., 646 (Den.)
Hull, R., Lovell, B. J., Openshaw, H. T., Payman, L. C. & Todd, A. R., (822) (Mal.)
Hummelen, L. R., 355 (Hel.)
Humphrey, A. A., 445 (Am.)
——, with Mirick, Zimmerman & Maner, 958 (Misc. Dis.)
Hundley, J. M. & Sebrell, W. H., 1061 (Def. Dis.)
Hunt, A. H., 1040 (Am.)
Hunter, E. A., 861 (Misc. Dis.)
Hunter, G. W., with Faust, Wright & McMullen, 756 (Hel.)
——, with Warren, V. G. & Warren, J., 1056 (Hel.)
——, Weller, T. H. & Jahnes, W. G., Jr., 788 (Ent.)
—— & Worth, C. B., 567 (Hel.)
Hunter, T. A. A., 293 (Mal.)
Huntress, R. L., with Peters & Porter, (346) (Hel.)
Husman, C. N., with Jones, Lindquist & Deonier (628) (Mal.)
——, with Lindquist, Madden & Travis, 795 (Ent.)
Hutchison, J. H., Pippard, J. S. & Gleeson-White, M. H., 338 (R.F.)
Huyck, J. H., with Stanbury, (257) (Ent.)
Hyder, G., with Karamchandani, 948 (Sp.)
Hyman, A. S., 155 (Misc. Dis.)
Hynd, R. S., 448 (R.F.)
Hynes, M., Ishaq, M. & Morris, T. L., 475 (Haem.)

I.

Iams, A. M., Tenen, M. M. & Flanagan, H. F., 587 (Der.)
Iaricci, V., with Mosto, 63 (Der.)
Ibarra Pérez, R. & González Prendes, M. A. 561 (Ys.), 563, 933 (Lep.)
——, with ——, 226 (Lep.)
d'Ignazio, C., 1042 (R.F.), 1130 (Typh.)
—— & Codeleoncini, E., 732, 1130, 1131 (Typh.), 1042 (R.F.)
Imbrosciano, G., 732 (Typh.)
Imperial Chemical (Pharmaceuticals) Limited, 1010 (Mal.)
India, 72, 968 (Reports, etc.), 81 (B.R.)
Indian Med. Gaz., 822 (Mal.)
Indian Research Fund Association, 129, 130, 131 *bis* (Chl.)
Ingle, L., with Kearns & Metcalf, 681 (Ent.)
Ingraham, H. S., with Zarafonetis & Berry, 746 (R.F.)
Innes, J., with Fenton, 298 (Mal.)
International Labour Review, 1082 (Reports, etc.)
Ionesco-Mihaiesti, C. & Ciuca, M., 321 (Typh.)
Iriarte, D. R., 1128 (Leish.)
Irigoyen Ramirez, A., with Romeo Viamonte, (197) (Mal.)
Irons, E. N., 734 (Typh.)
—— & Armbrust, C. A., Jr., 550 (Typh.)
Irons, J. V., Topping, N. H., Shepard, C. C. & Cox, H. R., 924 (Typh.)
Isaac, F., 652 (Am.)
Ishaq, M., with Hynes & Morris, 475 (Haem.)
Israels, M. C. G., with Wilkinson & Fletcher, 1065 (Haem.)

J.

Jackson, C. H. N., 312, 420 (Tryp.)
Jackson, E. B., with Smadel & Rights, 547, 919 (Typh.)
Jackson, W. P. U., 1169 (Der.)
Jacobs, H. R., 206 (Mal.)
Jacoby, H., 1187 (Misc. Dis.)
Jaffe, E., with Moore, Kessel, Simonsen, Marmorston, Llewellyn, Kaplan, Golden & Anderson, (650) (Dys.)
Jaffé, R., 350 (Hel.)
Jahn, F., with Johnson & Trussell, 67 (Prot.)
Jahnes, W. G., Jr., with Hunter & Weller, 788 (Ent.)
Jailer, J. W., 11 (Mal.)
Jalili, M., with Halawany, 540 (Leish.)
James, S. P., 783 (Misc. Dis.)
Jansen, G., 938 (Hel.)
Jaqueti Del Pozo, G., (935) (Lep.)
Jarcho, S., 3 (Mal.)
Jarvis, J. F., 224 (R.F.)
Jayewickreme, S. H., 966 (Ent.)
—— & Niles, W. J., 1079 (Ent.)
Jeffery, G. M., with Metcalf, Hess, Smith & Ludwig, 102 (Mal.)
Jellison, W. L., 31 (Typh.)
——, with Mackie, Davis, Fuller, Knapp, Steinacker, Stager, Traub, Millspaugh, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Jennings, G. H., 623 (Mal.)
Jensenius, H., 971 (B.R.)
Johnson, A. S., Jr. & Berry, M. G., 351 (Hel.)
Johnson, B. C., Mitchell, H. H. & Hamilton, T. S., 591 (Heat Str.)
——, with Shields, Hamilton & Mitchell, 591 (Heat Str.)
Johnson, C. M., with Shrapnel & Sandground, 928 (Am.)
Johnson, D. H. & Wharton, G. W., 640 (Typh.)
Johnson, G., Trussell, M. & Jahn, F., 67 (Prot.)
——, with ——, (254) (Prot.)
Johnson, H. A. & Eason, J. L., Jr., 1111 (Mal.)
——, with Parker, 13 (Mal.)
Johnson, H. C., with Darby & Jones, 667, 770 (Sp.)
Johnson, H. N., 194 (Rab.)
Johnson, J. A., Jr., Martin, W. B. & Breslow, L., 645 (Den.)
Johnson, M. B. & Damon, S. R., 915 (Typh.)
Johnson, M. S., 78 (Reports, etc.)
Johnstone, I. L., 1183 (Oph.)
Jokl, E., with Cluver & Rorich, (1084) (Reports, etc.)
Jones, E., with Darby, 949 (Sp.)
——, with —— & Johnson, 667, 770 (Sp.)
Jones, E. R., with Bell, (1123) (Tryp.)
Jones, H. A., with Deonier, 531 (Mal.)
——, ——, Burrell, R. W. & Knipling, E. F., 530 (Mal.)
——, with ——, Maple, Hinchey & Eide, 101 (Mal.)
——, Lindquist, A. W., Deonier, C. C. & Husman, C. N., (628) (Mal.)
——, with ——, Travis, Madden & Schroeder, 159 (Ent.)
Jones, J., with Dobes, 672 (Der.)
Jones, R., with Heidelberger, Mayer, Alving, Craige, Pullman & Whorton, 1013 (Mal.)
Jörg, M. E., with Mazza & Miyara, 208 (Tryp.)
Jorge, J. M. & Re, P. M., (1048) (Hel.)
J. Amer. Med. Ass., 301, 708 (Mal.)
Josey, A. I., with Leede, 573 (Hel.)
Joubert, F. J., with Polson & Haig, (954) (Vms.)
Journe, H., 15 (Bl.)
Joyce, S. J., with Sulzberger, Addenbrook, Greenberg & Mack, 1166 (Der.)
Joyner, L., with Fulton, 435 (Typh.)
Julião, O. F., 265 (B.R.)
Jung Sun, C., (721) (Leish.)
Jurkat, E., with Notestein, (261) (Reports, etc.)

K.

Kalandadze, L. P. & Sagatelova, I. S., 1106 (Mal.)
Kalmbach, E. R., 331 (Pl.)
Kamel, A., 1126 (Leish.), 1154 (Hel.)
Kane, C. A., with Most, London, Lavietes, Schroeder & Hayman, 1011 (Mal.)
Kan Huai-Chieh, (286) (Mal.)
Kaplan, A., 296 (Mal.)

Kaplan, F., with Moore, Kessel, Simonsen, Marmorston, Llewellyn, Golden, Anderson & Jaffe, (650) (Dys.)
Kaplan, L. I., Read, H. S. & Becker, F. T., 1110 (Mal.)
Kaplan, M. & Gluck, A. C., 438 (Y.F.)
Karamchandani, P. V. & Hyder, G., 948 (Sp.)
Kark, S. L. & Kark, E., 162 (Reports, etc.)
Kark, W., 559 (Am.), 780 (Heat Str.)
Karmann, G., with Arantes & Bier, (670) (Vms.)
Karnaky, K. J., 862 (Prot.)
Kartman, L., 740 (Pl.), 760 (Hel.)
——, Campau, E. J., Newcomb, E. H. & Morrison, F. D., 716 (Tryp.)
Katzenellenbogen, I., (597) (Misc. Dis.)
Kauer, G. L., Jr., with Lippard, 526 (Mal.)
Kauer, G. L., with Tompsett, 45 (Ys.)
Kauffmann, G., with Nitti & Conge, 749 (R.F.)
Kauntze, W. H., 1198 (Reports, etc.)
Kay, H. B., with Sangster, 29 (Typh.)
Kean, B. H., 867 (Reports, etc.)
—— & Grocott, R. G., 158 (Prot.)
—— & Tucker, H. A., 1189 (Misc. Dis.)
——, —— & Miller, W. C., 597 (Misc. Dis.)
Kearns, C. W., Ingle, L. & Metcalf, R. L., 681 (Ent.)
Keele, K. D., 1063 (Sp.)
—— & Bound, J. P., 368 (Sp.)
Keener, G. G., Jr., 197 (Mal.)
Keil, H., with Bechelli & Rotberg, 225 (Lep.)
Kekwick, A., 783 (Misc. Dis.)
——, with Cullinan, Watts & Titman, 942 (Def. Dis.)
Kelsey, F. E., with Oldham, 11 (Mal.)
——, —— & Geiling, E. M. K., 404 (Mal.)
——, ——, Cantrell, W. & Geiling, E. M. K., (625) (Mal.)
Kenamore, B., with Tsuchiya, 743 (Am.)
Kenney, M., 384 (Ent.)
Kern, F., Jr., 42 (Am.)
Kershaw, W. E., 557 (Am.)
Kervran, P., with Catanei, 247 (Der.)
——, with Lefrou, Loudoux & Le Poncin, 554 (Chl.)
Kessel, J. F., with Moore, Simonsen, Marmorston, Llewellyn, Kaplan, Golden, Anderson & Jaffe, (650) (Dys.)
Keys, A., with Mickelsen & Caster, 1061 (Def. Dis.)
Khairallah, A. A., with Hitti, 1137 (Den.)
Khalil Bey, M., Halawani, A. & Gamal Nor El Deen, 50 (Hel.)
Khartulari, E. M., with Alpatov & Nastukova, 69 (Ent.)
Kibbe, F. W., with Lewis, 304 (Mal.)
Kierland, R. R., Sheard, C., Mason, H. L. & Lobitz, W. C., 1008 (Mal.)
Kiker, C. C. & Sparkman, R. E., 308 (Mal.)
King, E. J., Gilchrist, M. & Tarnoky, A. L., (707) (Mal.)
——, Wootton, I. D. P. & Gilchrist, M., 822 (Mal.)
King, E. S., with Davis & Harrell, (143) (Hel.)
King, H., with Andrewes & Walker, 427, 728 (Typh.)
King, J. D., with Adams, Maegraith, Townshend, Davey & Havard, 402 (Mal.)
King, J. D., with Adams & Townshend, 400 (Mal.)
——, with Maegraith, Adams, Havard & Millet, 238 (Sp.)
——, with ——, ——, Tottey, Rigby & Sladden, 821 (Mal.)
——, with ——, ——, Townshend, Davey & Havard, 402 (Mal.)
King, W. D. & Sebrell, W. H., 854 (Def. Dis.)
Kinsey, F. R., with Herbut, 959 (Misc. Dis.)
Kirby, H., 222 (Am.), 254 (Prot.)
Kirby-Smith, H. T., 148 (Vms.)
Kirk, R. & Lewis, D. J., 796, 1195 (Ent.)
Kirk, R. L., with Gunn & Waterhouse, 502 (Reports, etc.)
Kirman, B. D., 944 (Def. Dis.)
Kiskaddon, R. M. & Renshaw, R. J. F., 158 (Prot.)
Kitchen, S. F., with Boyd, 304, 1003 (Mal.)
—— & Sadler, G. G., 415 (Bl.)
Kleeberg, J., 393 (Mal.)
Klein, E. F., with Browning, Raphael & Coblenz, 548 (Typh.)
Klein, H. S., 120 (Typh.)
Kleine, F. K., 259 (Reports, etc.)
Kligman, A. M., with Weidman, 778 (Der.)
Kline, B. S., with Suessenguth, 360 (Hel.)
Klingensmith, C. W., 648 (Pl.)
Knapp, J. A., with Mackie, Davis, Fuller, Steinacker, Stager, Traub, Jellison, Millspaugh, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Knight, K. L., with Belkin & Rozeboom, 6 (Mal.)
——, with Rozeboom, 817 (Mal.)
Knipe, F. W., 1191 (Ent.)
Knipling, E. F., 493 (Ent.)
——, with Jones, Deonier & Burrell, 530 (Mal.)
——, with Lindquist, Madden & Wilson, 159 (Ent.)
——, with Madden & Lindquist, 158 (Ent.)
Knisely, M. H., Eliot, T. S. & Bloch, E. H., 631 (Mal.)
——, Stratman-Thomas, W. K., Eliot, T. S. & Bloch, E. H., 1113 (Mal.)
Knoll, E. W. & Howell, K. M., 444 (Am.)
Knott, J., with Carrión, 246 (Der.)
Knowles, F. L. & Fisk, F. W., 529 (Mal.)
—— & Smith, C. S., 710 (Mal.)
Knox, W. E., 1065 (Mal.)
Koch, M. B., with Spies, Lopez, Menendez & Minnich, 474 (Sp.)
——, with ——, Milanes, Menéndez & Minnich, 769 (Sp.)
——, with ——, Vilter & Caldwell, 375 (Haem.)
——, with Vilter & Spies, 375 (Haem.)
Koch, W., 594 (Heat Str.)
Kocharian, A. A., with Ananian, Chubkova & Azizian, 13 (Mal.)
Koenigsfeld, E. G. H., 191 (Rab.)
Koepfli, J. B., with Mead, Rapport, Senear & Maynard, 823 (Mal.)
Koepke, J. A., with Rumreich, 429 (Typh.)
Kohls, G. M., with Blake, Maxcy, Sadusk & Bell. 327 (Typh.)

Kohls, with Cooley, 864 (Ent.)
——, with Mackie, Davis, Fuller, Knapp, Steinacker, Stager, Traub, Jellison, Millspaugh, Austrian, Bell, Wei Hsi & Girsham, 917 (Typh.)
——, with Philip, 119 (Typh.)
Kojevnikov, P. V., 724 (Leish.)
Kopciowska, L., with Silva, 428 (Typh.)
Kordy, M. I., with Halawani, (1143) (Am.)
Kornblueth, W., with Feigenbaum, 1074 (Misc. Dis.)
Krakower, C., Hoffman, W. A. & Axtmayer, J. H., 227 (Hel.)
Krapcho, J., with English, Clark, Shepherd, Mason & Roblin, (905) (Mal.)
Kraus, H., 869 (B.R.)
Kringelbach, J., with Frandsen, 1163 (Sp.)
Krishnan, B. G., Ramachandran, S. & Sadhu, K., 473 (Def. Dis.)
Kruse, C. W. & Gartrell, F. E., (308) (Mal.)
—— & Metcalf, R. L., 1111 (Mal.)
Kuffler, S. W., with Harvey & Tredway, 490 (Misc. Dis.)
Kuitunen-Ekbaum, E., 940 (Hel.)
Kumm, H. W., Osborno-Mesa, E. & Boshell-Manrique, J., 599 (Ent.)
Kutzenok, A., with Olitzki, Oleinik & Czaczkes, (635) (Typh.)
Kuzmina, L. A., with Pavlovsky, 744 (R.F.)
Kuznetzov, B., with Bobrinskoy & Kuzyakin, 268 (B.R.)
Kuzyakin, A., with Bobrinskoy & Kuznetzov, 268 (B.R.)
Kwoh, C. S., (286) (Mal.)
Kyker, G. C., Cornatzer, W. E. & McEwen, M. M., 706 (Mal.)

L.

Lacaz, C. da S., (488) (Der.)
Lackey, J. B. & Steinle, M. L., 794 (Ent.)
Ladell, W. S. S., 250, 675 (Heat Str.)
Laemmert, H. W., Jr., 644 (Y.F.)
—— & de Castro Ferreira, L., 33 (Y.F.)
Lagodsky, H., with Launoy, (1025) *bis* (Tryp.)
Lagos, Nigeria, 838 (Y.F.)
Laha, P. N., 9, (394) (Mal.)
Lahiri, M. N., 137 *bis* (R.F.)
Lahnum, W. H., 741 (Pl.)
Laigret, J. & Corcos, A., 739 (Den.)
Lairet, F., Jr., 437 (Y.F.)
de Lajudie, P., with Floch, 342, 933 (Lep.), 352, (852), 1155 (Hel.), 704 (Mal.), 908 (Tryp.)
Lambert, J. D., 203 (Mal.)
Lampson, R. S., with Gilje, (444) (Am.)
Lamy, L., 442, 743 (Am.)
——, with Deschiens, (352) (Hel.)
Lancet, 416 (Bl.), 485 (Der.)
Landsborough, D., (1158) (Hel.)
Lane, J. D., with Binford, 487 (Der.)
Lane, W. F., with Day & Wood, (853) (Hel.)
Láng, S., 43 (Am.)
Lange, K. & Matzner, M. J., 1109 (Mal.)
Laptev, A. A., 50 (Hel.)
Larcher, M. & Romaüa, C., 110 (Tryp.)
Larenas, M., with León & Beltrán, 1017 (Mal.)
Larsen, E. B. & Thomsen, M., 1194 (Ent.)
Larsh, J. E., Jr., (351), 759 *bis* (Hel.)
Lasch, W., with Gruenfelder, 1148 (Lep.)
Lasnier, E. P. & Cassinelli, J. F., 567 (Hel.)
Latif, N. & El Kordy, M. I. (1158) (Hel.)
Latyshev, N. I., 602 (Ent.)
Laube, P. J., with Yang, 661 (Hel.)
Launoy, L., 314, 423, 424 (Tryp.)
—— & Lagodsky, H., (1025) *bis* (Tryp.)
Laurent, A., with Florio, Hammon & Stewart, 737 (Den.)
Laus, F., with Ramos, 909 (Tryp.)
Lauterburg-Bonjour, M., (494) (Ent.)
Lavier, G. & Stefanopoulo, G., 463 (Hel.)
Lavietes, P. H., with Most, London, Kane, Schroeder & Hayman, 1011 (Mal.)
Lavoipierre, M., with de Meillon, (160) (Ent.)
Lawless, D. K., 651 (Am.)
Lawrence, E. A., with Abbey, 627 (Mal.)
—— & Bennett, R. E., 36 (Dys.)
Lawton, A. H., with Ashburn, Perrin & Brady, 662 (Hel.)
——, Brady, F. J., Ness, A. T. & Haskins, W. T., 55 (Hel.)
League of Nations, 268 (B.R.)
Lebied, B., with Wanson, 1191 (Ent.)
Le Chuiton, F., (625) (Mal.)
—— & Berge, C., 915 (Typh.)
Leckie, M. W., with Browning, Calver & Walls, 633 (Tryp.)
Lee, R. K. C. & Pang, H. Q., 66 (Misc. Dis.)
Leede, W. E. & Josey, A. I., 573 (Hel.)
Lefrou, G., Kervran, P., Loudoux, Y. & Le Poncin, N., 554 (Chl.)
Le Gac, P., 212, 916 *bis*, (917) (Typh.)
—— & Borjeix, L., (327) (Typh.)
——, Seite, P. & Combescot de Marsaguet, G., 621 (Mal.)
Le Gall, R., 284 (Mal.)
Lehmann, G. D. & Prendiville, J. T., 787 (Prot.)
Leishman, A. W. D., 366 (Sp.)
Leitão, J. S., 1127 (Leish.)
Leitman, M. Z., 1139 (Am.)
—— & Vitlinskaya, I. A., 1143 (Am.)
Leitner, A. J., 381 (Misc. Dis.)
Lendrum, A. C., with Fisher, 1186 (Misc. Dis.)
Lengauer, L., 459 (Lep.)
Lennhoff, L., with van den Ende, Locket, Hargreaves & Niven, 918 (Typh.)
León, A. P., 731 (Typh.)
——, Beltrán, E. & Larenas M., R., 1017 (Mal.)
—— & Cano, C., 731 (Typh.)
Leon, L. A., 1059 (Hel.)
Leon Blanco, F., with Oteiza, 587 (Der.)
Leon y Blanco, F. & Sanchez Garcia, E., 586 (Der.)
Leonova, N. A., 746 (R.F.)
——, with Sofiev, 744 (R.F.)
Le Poncin, N., with Lefrou, Kervran & Loudoux, 554 (Chl.)
Leprosy Review, 461 (Lep.)
Lerner, E. M., 1081 (Lab.)
Le Roy, with Martin, Sureau, Babouot & Bourcart, 464 (Hel.)
Leslie, A., with Silverman, 39 (Am.)
Levaditi, J. C. & Panthier, R., 731 (Typh.)
Levi-Castillo, R., (7) (Mal.)
Levine, A. C., with Gottfried, 301 (Mal.)
Levy, B. M., 150 (Der.)

Lewillon, R., 208 (Tryp.)
——, with van Hoof, Henrard, Peel & Rodjestvensky, 536 (Tryp.)
Lewis, B. O., with Sodeman, 135 (Am.)
Lewis, D. J., with Kirk, 796, 1195 (Ent.)
Lewis's Medical, Scientific & Technical Library, 270 (B.R.)
Lewis, R. A. & Kibbe, F. W., 304 (Mal.)
——, with Senekjie, 320 (Leish.), 677 (Prot.)
Lherisson, C., with Augustine, 570 (Hel.)
Lidz, T., with Tumulty, Nichols & Singewald, 819 (Mal.)
Liebermann, H. R., 334 (Am.)
Liebig, H., with Vollmer, 201 (Mal.)
Liem, S. D. & van Thiel, P. H., 719 (Tryp.)
Liger & Schneyder, R., (939) (Hel.)
Likoff, W., 1133 (Typh.)
Lillie, R. D., 603 (Lab.)
Lima, L. de S. & Cerqueira, G. de C., 47, 226 (Lep.)
Lindberg, D. O. N., with Bayley & Baggenstoss, (597) (Misc. Dis.)
Lindquist, A. & McDuffie, W. C., 795 (Ent.)
Lindquist, A. W., with Gahan, 99 (Mal.)
——, with —— & Travis, 99 (Mal.)
——, with ——, —— & Morton, 99 (Mal.)
——, with Jones, Deonier & Husman, (628) (Mal.)
——, Madden, A. H., Husman, C. N. & Travis, B. V., 795 (Ent.)
——, with —— & Knipling, 158 (Ent.)
——, ——, Wilson, H. G. & Knipling, E. F., 159 (Ent.)
——, with Schroeder, 963 (Ent.)
——, Travis, B. V., Madden, A. H., Schroeder, H. O. & Jones, H. A., 159 (Ent.)
——, Wilson, H. G., Schroeder, H. O. & Madden, A. H., 159 (Ent.)
Lindsay, A., with Bovarnick & Hellerman, 824, 825 (Mal.)
Lindsay, S. F., 786 (Misc. Dis.)
Lindskog, G. E. & Walters, W., 1140 (Am.)
Lipman, M. O., with Rose & Culbertson, 232 (Hel.)
Lippard, V. W. & Kauer, G. L., Jr., 526 (Mal.)
Lippincott, S. W., with Ellerbrook, Cateno, Gordon & Marble, 626 (Mal.)
——, ——, Hesselbrock, W. B., Carrico, C. C. & Marble, A., 1009 (Mal.)
——, ——, ——, Gordon, H. H., Gottlieb, L. & Marble, A., 523 (Mal.)
——, with Goodman, Weinberger, Marble & Wright, 573 (Hel.)
——, with Gordon, Marble, Engstrom & Brunsting, 1007 (Mal.)
Litchfield, J. T., Jr., with Marshall & White, 826 (Mal.)
Little, P. A. & Subbarow, Y., 538 (Tryp.)
Liu, W. T., with Snyder & Zarafonetis, 426 (Typh.)
Liu, Y., with Tan, 333 (Am.)
Livadas, G. A., (1015) (Mal.)
Livesay, H. R., with Pollard, Wilson & Woodland, 738, 925 (Den.)
Llewellyn, H., with Moore, Kessel, Simonsen, Marmorstan, Kaplan, Golden, Anderson & Jaffe, (650) (Dys.)
Lobitz, W. C., with Kierland, Sheard & Mason, 1008 (Mal.)
Lobo, R., 938 (Hel.)
Locket, S., 417 (Bl.)
——, with van den Ende, Hargreaves, Niven & Lennhoff, 918 (Typh.)
Loeb, P., with Richardson, Walker & Miller, 446 (R.F.)
Loeb, R. F., 708 (Mal.)
Löffler, W., with Mooser, 543 (Typh.)
Lofgren, R. & Soule, M. H., 335, (446) (R.F.)
London, I. M., with Most, Kane, Lavietes, Schroeder & Hayman, 1011 (Mal.)
Lopes, C. F., 562 (Ys.)
Lopez, G. G., with Spies, Menendez, Minnich & Koch, 474 (Sp.)
López-Fernández, J., with Talice, 112 (Tryp.)
Lopez Toca, R., with Spies, Frommeyer, Garcia Lopez & Gwinner, 949 (Sp.)
Loranger, G. L., with Woodward & Philip, 836 (Typh.)
Lorentz, F. H., 117 (Typh.)
Loudoux, Y., with Lefrou, Kervran & Le Poncin, 554 (Chl.)
Lourie, E. M., with Collier, 1126 (Leish.)
——, with Williamson, 845 (R. F.)
Lovell, B. J., with Hull, Openshaw, Payman & Todd (822) (Mal.)
Lovibond, J. L., 1040 (Am.)
Low, G. C., 317 (Leish.), 783 (Misc Dis.)
Lowe, J., 523 (Mal.), 540 (Leish.), (867) (Reports, etc.), 1129 (Typh.)
——, with Das Gupta & Chakravarti, 298 (Mal.)
Lozano Morales, A., 205 (Mal.)
Lucas, R. B., 651 (Am.)
Luckiesh, M., Taylor, A. H., Cole, H. N. & Sollmann, T., 675 (Heat. Str.)
Ludwig, G. W., with Metcalf, Hess, Smith & Jeffery, 102 (Mal.)
Lupascu, G., (817) (Mal.)
Lusk, J. W., 426 (Typh.)
Luthra, P. N., 1184 (Ulc.)
Lwoff, M., Bovet, D. & Funke, A., 313 (Tryp.)
—— & Nicolle, P., (1026) (Tryp.)
Lyman, F. E., with Price, 14 (Mal.)

M.

Macan, T. T., 203 (Mal.)
McArthur, J. N., 1000 (Mal.)
MacArthur, W., (903) (Mal.)
Mac Arthur, W. P., 602 (Ent.)
McCall, K. B., with Cooperman, Elvehjem & Ruegamer, 772 (Haem.)
MacCallum, F. O., with Buckland, Dudgeon, Edward, Henderson-Begg, Niven, Rowlands & van den Ende, Bargmann, Curtis & Shepherd, 436 (Typh.)
MacCallum, W. P., 516 (Mal.)
McCarthy, D. D., with Dick, 660 (Hel.)
Macchiavello, A., 647 (Pl.)
—— & Cifuentes, O., 23 (Typh.)
——, ——, & Ovalle, H., 1032, 1033 (Typh.)
—— & Ovalle, H., 1033 (Typh.)
McCollum, W. T., with Frachtman, 493 (Misc. Dis.)
McCoy, O. R., 1073 (Misc. Dis.)
McCullagh, McK., 783 (Misc. Dis.)
McCulloch, R. N., 1034 (Typh.)

McDaniel, F. L., White, B. V., Jr. & Thompson, C. M., 1060 (Def. Dis.)
Macdonald, G., 684 (Reports, etc.)
McDonald, S., 193 (Rab.)
McDuffie, W. C., with Lindquist, 795 (Ent.)
McEwen, M. M., with Kyker & Cornatzer, 706 (Mal.)
McGovern, V., 30 (Typh.)
McGovran, E. R., Fales, J. H. & Goodhue, L. D., 962 (Ent.)
Macgregor, I., 1138 (Dys.)
McIntosh, D. G., with Brodie, Cook & Drysdale, 841 (Dys.)
Mack, A. G., with Sulzberger, Addenbrooke, Joyce & Greenberg, 1166 (Der.)
Mackay-Dick, J., 128 (Pl.)
Mackie, T. T. *et al.*, 1132 (Typh.)
——, Davis, G. E., Fuller, H. S., Knapp, J. A., Steinacker, M. L., Stager, K. E., Traub, R., Jellison, W. L., Millspaugh, D. D., Austrian, R. C., Bell, E. J., Kohls, G. M., Wei Hsi & Girsham, J. A. V., 917 (Typh.)
McKibbin, J. M., with Williamson, Hegsted, & Stare, 1009 (Mal.)
McKinney, W. W., with Robinson, 298 (Mal.)
Mackinnon, J. E., with Talice, 292 (Mal.)
Mackinnon, P. G., with Moyne, Stubbs, Crowdy, Citrine, Blacklock, Engledow & Henderson, 70 (Reports, etc.)
Mackworth, N. H., 1071 (Heat Str.)
Macleod, J., with Craufurd-Benson, 638 (Typh.)
McLetchie, J. L., 1022 (Tryp.)
MacLouf, A. C., with Brumpt, 835 (Typh.)
McMahon, M. C., with Wayson, 648 (Pl.)
McMartin, W. J., 231 (Hel.)
McMullen, D. B. & Beaver, P. C., 138 (Hel.)
——, with Faust, Wright & Hunter, 756 (Hel.)
McNaught, J. B., (355), (362) (Hel.)
Macnaught, W. W., 391 (Mal.)
Madden, A. H., with Lindquist, Husman & Travis, 795 (Ent.)
——, —— & Knipling, E. F., 158 (Ent.)
——, with ——, Travis, Schroeder & Jones, 159 (Ent.)
——, with ——, Wilson & Knipling, 159 (Ent.)
——, with ——, —— & Schroeder, 159 (Ent.)
Madinaveitia, J., 1011 (Mal.)
Maegraith, B. G., *et al.*, 99 *bis*, 416, 905 (Mal.), 553 (Y.F.)
——, Adams, A. R. D., Havard, R. E., King, J. D. & Millet, R. F., 238 (Sp.)
——, ——, King, J. D., Tottey, M. M., Rigby, D. J. & Sladden, R. A., 821 (Mal.)
——, ——, ——, Townshend, R. H., Davey, T. H. & Havard, R. E., 402 (Mal.)
——, with ——, ——, ——, —— & ——, 402 (Mal.)
——, with Spinks & Tottey, (707) (Mal.)
Magalhães, B. F. & Freire, S. A., (1123) (Tryp.)
Magath, T. B. & Mathieson, D. R., 350, 755, 756 (Hel.)
Magnuson, H. J., with Schatz, Waksman & Eagle, 1025 (Tryp.)
Magoon, E. H., 877 (B.R.)
Magrou, E., 926 (Pl.)
—— & Brisou, J., 926 (Pl.)
Magy, H. I. & Hoskins, W. M., 601 (Ent.)
Mahaffy, A. F., with Smithburn, 34 (Y.F.)
Mahfouz, M., with Amin & Sherif, 43 (Am.)
Major, F. W., with Barber & Wragg, (1011) (Mal.)
Majumder, A. R., Bagchi, A. K. & Ghosh, B. K., 441 (Dys.)
Makari, J. G., 623 (Mal.)
Malanga, C., with Seeler & Pierson, 309 (Mal.)
Malbrant, R., with Parrot, (160) (Ent.)
Malcomson, M. E., with Craigie, Watson & Clark, 914 (Typh.)
Maldonado, J. F., 1143 (Am.)
Maldonado Capriles, J., (1195) (Ent.)
Malo-Juvera, F., with Ortiz-Mariotte & Payne, 324 (Typh.)
Mandekos, A. G., 906 (Mal.)
Mandelstam, J., with Gillman & Gillman, 666 (Def. Dis.)
Maner, G. D., with Mirick, Zimmerman & Humphrey, 958 (Misc. Dis.)
Manson-Bahr, P., 317 (Leish.), 607 (B.R.) 684, 688, 1199, 1200 (Reports, etc.), 783 1072 (Misc. Dis.)
Manson-Bahr, P. E. C. & Charters, A. D., 1186 (Misc. Dis.)
Manson, J. M. S., 1202 (Reports, etc.)
Manuwa, S. L. A., 782 (Ulc.)
Manwell, R. D., 495 (Lab.)
Maple. J. D., with Deonier, Burrell & Cochran, 101 (Mal.)
——, with ——, Jones, Hinchey & Eide, 101 (Mal.)
Mara, L., 1078 (Ent.)
Marble, A., with Ellerbrook, Lippincott, Cateno & Gordon, 626 (Mal.)
——, with Goodman, Weinberger, Lippincott & Wright, 573 (Hel.)
——, with Gordon, Engstrom, Brunsting & Lippincott, 1007 (Mal.)
——, with Lippincott, Ellerbrook, Hesselbrock & Carrico, 1009 (Mal.)
——, with ——, ——, ——, Gordon & Gottlieb, 523 (Mal.)
Mariani-Tosatti, G., 49 (Hel.)
Mariano, J., 46 (Lep.)
Marill, F. G. & Alcay, L., (354) (Hel.)
Mario, S., (524) (Mal.)
Markowitz, J., 741 (Chl.)
Markson, J. L. & Dawson, J., 12 (Mal.)
Marliangeas, Mlle., with Montel & Brun, Mlle., 454 (Lep.)
Marmorston, J., with Moore, Kessel, Simonsen, Llewellyn, Kaplan, Golden, Anderson & Jaffe, (650) (Dys.)
Marneffe, H., with Sautet, (258) (Reports, etc.), 462 (Hel.)
Marple, C. D., (377) (Der.)
Marques, A. P., with Bancroft, Guinto & Rodriguez, 454 (Lep,)
Marray, A. M. & Philipzs, G. C. L., (136) (Am.)
Marshall, E. K., Jr., (905) (Mal.)
——, with Dearborn, 404 (Mal.)
——, Litchfield, J. T. & White, H. J., 826 (Mal.)

Marshall, P. B., 15, 97 (Mal.)
——, with Goodwin, 315 (Tryp.)
—— & Rogers, E. W., 98 (Mal.)
Marson, H. W., with English, Clark, Shepherd, Krapcho & Roblin, (905) (Mal.)
Martin, R., Le Roy, Sureau, B., Badouot, P. & Bourcart, N., 464 (Hel.)
——, with Parrot, (601) (Ent.)
Martin, W. B., 669 (Vms.)
——, with Johnson & Breslow, 645 (Den.)
Martínez Báez, M. & Villasana, A., 747 (R.F.)
Martins, A. V., 776 *bis* (Vms.)
—— & Versiani, V., (904) (Mal.)
——, with —— & Sobrinho, 754 (Hel.)
——, —— & Tupinambá, A. A., 718 *bis* (Tryp.)
Maruashvili, G. M., 38 (Am.), 45 (R.F.)
Marvin, H. N. & Rigdon, R. H., 715 (Mal.)
Mason, H. L., with Kierland, Sheard & Lobitz, 1008 (Mal.)
Mason, P. K., Daniels, W. B., Paddock, F. K. & Gordon, H. H., 1157 (Hel.)
Mathieson, D. R., with Magath, 350, 755, 756 (Hel.)
Mathur, W. & Goyal, R., 217 (Pl.)
Matzner, M. J., with Lange, 1109 (Mal.)
Maxcy, K. F., with Blake, Sadusk, Kohls & Bell, 327 (Typh.)
Mayer, J. H., 156 (Misc. Dis.)
Mayer, M., with Heidelberger, Alving, Craige, Jones, Pullman & Whorton, 1013 (Mal.)
Mayer, M. M., with Heidelberger & Coates, 1013 (Mal.)
——, with —— & Demarest, 816 (Mal.)
Mayer, R. L. & Brousseau, D., 1125 (Tryp.)
Maynard, J. T., with Mead, Rapport, Senear & Koepfli, 823 (Mal.)
Mazza, S., 831 (Tryp.)
——, Basso, G. & Basso, R., 720, 909 (Tryp.)
——, Miyara, S. & Jörg, M. E., 208 (Tryp.)
Mazzotti, L., 137 (Hel.)
—— & Osorio, M. T., 472 (Hel.)
—— & Varela, G., 1135 (Typh.)
Mbappe, J., with Chabeuf, 1122 (Tryp.)
Mead, J. F., Rapport, M. M., Senear, A. E., Maynard, J. T. & Koepfli, J. B., 823 (Mal.)
Mears, A. R. R., 498 (Reports, etc.)
Méchin, R., 1127 (Leish.)
Medical Advisory Division, Headquarters, Supreme Allied Command, S.E. Asia, 203 (Mal.)
Medical J. Australia, 529 (Mal.)
Megaw, J., 603 (Reports, etc.)
de Meillon, N. & Davis, D. H. S., 966 (Ent.)
—— & Lavoipierre, M., (160) (Ent.)
de Meira, B. T. V., with da Cruz Ferreira, 863 (Ent.)
——, Girão, J. & Coito, A. de M. F. (760) (Hel.)
Meleney, H. E., (223) (Am.)
——, with Zuckerman, 223 (Am.)
Mellanby, K., 1132 (Typh.)
Mello, G. B., with Causey, 287 (Mal.)
de Mello, J. P., (820) (Mal.)
de Mello, R. P., with Cruz, 660 (Hel.)
Melville, A. R., Wilson, D. B., Glasgow, J. P. & Hocking, K. S., 92 (Mal.)
Mendell, T. H., 548 (Typh.)
Mendelsohn, H. V., 673 (Der.)
Méndez, T. H., with Del Pozo & González, (148) (Vms.)
Menéndez, A., with Spies, Milanes, Koch & Minnich, 769 (Sp.)
Menendez, J. A., with Spies, Lopez, Minnich & Koch, 474 (Sp.)
Menon, I. G. K., 595, 860 (Misc. Dis.)
Menon, T. B. & Veliath, G. D., (297) (Mal.)
Mergener, J. C., 405 (Mal.)
Merino, C., 552 (Bart.)
Merrill, D., with Rubenstein & Shulman, 292 (Mal.)
Merskey, C. & Baskind, E., 952 (Haem.)
Mersky, C., (493) (Misc.)
Mertens, E., 257 (Lab.)
Mesrobeanu, L., with Ciucă, (210) (Typh.)
——, with ——, Badenski, Franke & Munteanu, (210) (Typh.)
——, with ——, ——, Munteanu & Paraschivescu, (210) (Typh.)
Metcalf, R. L., 198, 392 (Mal.)
——, Hess, A. D., Smith, G. E., Jeffery, G. M. & Ludwig, G. W., 102 (Mal.)
——, with Kearns & Ingle, 681 (Ent.)
——, with Kruse, 1111 (Mal.)
de Meulenaere, J., with Vincke, Parent & Berteaux, 291 (Mal.)
Meyer-May, J., with Ho-Dac-Di. Vu-Dinh-Tung, Tong-That-Tung & Dinh-Van-Thang, 87 (B.R.)
Meyersburg, H. A., 59 (Def. Dis.)
Michael, P., 53 (Hel.)
Michaelson, I. C., with O'Donovan 1175 (Oph.)
Michener, C. D., 797, 965 (Ent.)
Mickelsen, O., Caster, W. O. & Keys, A., 1061 (Def. Dis.)
Milanes, F., with Spies, Menéndez, Koch & Minnich, 769 (Sp.)
Miles, V. I., (199) (Mal.)
Millan Gutierrez, J., 1128 (Leish.)
Miller, E. S. & Beeson, P. B., 732 (Typh.)
Miller, H., with Moseley, 316 (Tryp.)
Miller, I., with Richardson, Walker & Loeb, 446 (R.F.)
Miller, J. F., with Whittier & Einhorn, 577 (Hel.)
Miller, S. E., 49 (Hel.)
Miller, W. C., with Kean & Tucker, 597 (Misc. Dis.)
Millet, R. F., with Maegraith, Adams, Havard & King, 238 (Sp.)
Mills, W. G., 346 (Hel.)
Millspaugh, D. D., with Mackie, Davis, Fuller, Knapp, Steinacker, Stager, Traub, Jellison, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Milovzorova, E. N., 1003 (Mal.)
Milwidsky, H., 568 (Hel.)
Minnich, V., with Spies, Lopez, Menendez & Koch, 474 (Sp.)
——, with ——, Milanes, Menéndez & Koch, 769 (Sp.)
Minton, J., 1175 (Oph.)
Mirick, G. S., Zimmerman, H. M., Maner, G. D. & Humphrey, A. A., 958 (Misc. Dis.)
Missiroli, A., (827) (Mal.)

Mitchell, H. H., with Johnson & Hamilton, 591 (Heat Str.)
——, with Shields, Johnson & Hamilton, 591 (Heat Str.)
——, with Spector & Hamilton, (378), 591 *bis* (Heat Str.)
Mitchell, J. P., 78 (Reports, etc.)
Mitra, S. K., (857) Der.)
Mitra, S. N., with Bose, Ghosh & Datta, 1028 (Leish.)
Miyara, S., with Mazza & Jörg, 208 (Tryp.)
Mohan, B. N., 197 (Mal.)
Mohanty, J. K., 137 (R.F.), 250 (Ulc.), (380) (Misc. Dis.)
Mohr, W., 406, 408 (Mal.)
Mohun, A. F., 866 (Lab.)
Moiser, B., 454 (Lep.)
Moll. A. A., 505 (B.R.)
Mollaret, P. & Digo, R., (653) (R.F.)
Mom, A. M. & Basombrio, G., 455 (Lep.)
Monchadsky, A., with Blagoveschensky & Bregetova, (160) (Ent.)
Mondor, 87 (B.R.)
Monsanto Chemical Company, 648 (Pl.)
Montel, M. L. R., 503 (B.R.)
Montel, R., 344, 1152 *bis* (Lep.), 449, 450 (Ys.)
—— & Basset, (342) (Lep.)
——, Brun, Mlle. & Marliangeas, Mlle., 454 (Lep.)
Monteny, V. A. R., 1060 (Def. Dis.)
Montézin, G., with Funke & Bovet, 713 (Mal.)
Moore, C. V., Bierbaum, O. S., Welch, A. D., & Wright, 477 (Haem.)
Moore, D. F., 1180 (Oph.)
Moore, F. J., Kessel, J. F., Simonsen, D. G. & Marmorston, J., with Llewellyn, H., Kaplan, F., Golden, F., Anderson, N. & Jaffe, E., (650) (Dys.)
Moore, J. A., Young, M. D., Hardman, N. H. & Stubbs, T. H., 1002 (Mal.)
——, with ——, Stubbs, Ehrman, Hardman, Ellis & Burgess, 5 (Mal.)
Moore, L. D., 4 (Mal.)
Moore, M. & Ackerman, L. V., 957 (Der.)
Mooser, H., 611 (B.R.)
—— & Löffler, W., 543 (Typh.)
Morales, A. L., 634 (Leish.)
Morales, F. H., 837 (Typh.)
——, with Oliver-González, 471 (Hel.)
Moreau, P. & Ouary, G., 378 (Ulc.)
Morey, M., with Spies, 768 (Def. Dis.)
Morgan, C. N., 1040 (Am.)
Mornet, J., with Parrot & Cadenat, (160) (Ent.)
Mornet, P., with Parrot & Cadenat, (602) (Ent.)
Morris, K. R. S., 537 (Tryp.)
Morris, T. L., with Hynes & Ishaq, 475 (Haem.)
Morrison, F. D., with Kartman, Campau & Newcomb, 716 (Tryp.)
Morrissey, G. C. & Derrick, E. H., 31 (Typh).
Morton, F. A., with Gahan, Travis & Lindquist, 99 (Mal.)
——, with Snyder, 1196 (Ent.)
Moseley, V. & Miller, H., 316 (Tryp.)
Moss, E. S., with Burns & Brueck 468 (Der.)
Most, H., with Birnkrant & Greenberg, 221 (Am.)
—— & Hayman, J. M., Jr., 1004, 1005 (Mal.)
——, London, I. M., Kane, C. A., Lavietes, P. H., Schroeder, E. F. & Hayman, J. M., Jr., 1011 (Mal.)
Mosto, D. & Iaricci, V., 63 (Der.)
Mottoulle, L., (1202) (Reports, etc.)
Moulder, J. W. & Evans, E. A., Jr., 1118 (Mal.)
——, with Speck & Evans, 1116 (Mal.)
Moulton, F. R., 881 (B.R.)
Mourão, B. M., 585 (Der.)
Moustardier, G., with Cornil & Poursines, 216 (Pl.)
Moyne, Stubbs, R. E., Crowdy, R. E., Citrine, W., Mackinnon, P. G., Blacklock, M. G., Engledow, F. L. & Henderson, H. D., 70 (Reports, etc.)
Mugrage, E. R., with Florio & Stewart, 438 (Den.)
Muir, E., 458, 654 (Lep.)
——, with Rogers, 689 (B.R.)
Mukerjee, S., 1030 (Leish.)
Mukerji, A. K. & Sen Gupta, K. K., 230 (Hel.)
Mukerji, B., 1012 (Mal.)
——, with Dutta & Sekar, 1029 (Leish.)
Muller, H. E., with Chermock, 968 (Lab.)
Mullett, C. F., (196) (Rab.)
Munteanu, G., with Ciuca, Mesrobeanu, Badenski & Franke, (210) Typh.)
——, with ——, ——, —— & Paraschivescu, (210) (Typh.)
Munter, E. J. & Packchanian, A., 721 (Leish.)
Murgatroyd, F., 1040 (Am.)
Murphy, R. C., Jr. & Shapiro, S., 581 (Haem.)
Murray, E. S., with Ecke, Gilliam, Snyder, Yeomans & Zarafonetis, 545 (Typh.)
——, with Yeomans, Snyder, Ecke & Zarafonetis, 636 (Typh.)
——, with Zarafonetis, Ecke, Yeomans & Snyder, 912 (Typh.)
——, —— & Snyder, J. C., 433 (Typh.)
——, with —— & ——, 922 (Typh.)
Muspratt, J., 704, (1000) (Mal.)
Musselman, M. M., 664, 665 (Def. Dis.)
Muwazi, E. M. K., 747 (R.F.)
——, with Trowell, 143, 363, (Def. Dis.)

N.

Naftalin, J. M., with Bookless, 199 (Mal.)
Nagley, L., 296 (Mal.)
Nail, S. K., with Soman, 439 (Chl.)
Nájera Angulo, L., 721 (Leish.)
——, 447 (R.F.), (647) (Den.), 1026, 1027 *bis* (Leish.)
Nájera, L., 284 (Mal.), (317) (Tryp.), 832 (Leish.)
Napier, L. E., 10 (Mal.), 77, 684 (Reports, etc.), 783 (Misc. Dis.), 867 (B.R.)
Nashed, E., (1184) (Oph.)
Nastukova, O. K., with Alpatov & Khartulari, 69 (Ent.)
Nath, T., 1184 (Ulc.)
Naval Medical Research Institute and U.S. Naval Hospital (1157) (Hel.)
Nayar, S. L., with Badhwar & Chopra, 672 (Der.)

Neel, R., (313) (Tryp.)
Neff, F. C., with Davis, 779 (Der.)
Neghme R., A., 577 (Hel.)
Nelson, A. A., with Fitzhugh, Calvery & Glassman, 525 (Mal.)
Nelson, E. C. & Bayliss, M., 1046 (Hel.)
Nelson, J. W., 535 (Tryp.)
Nelson, L. M., 303 (Mal.)
Nery Guimarães, F., 381 (Prot.)
Nesbitt, H. H. J., (864) (Ent.)
Ness, A. T., with Lawton, Brady & Haskins, 55 (Hel.)
Nettel F., R., 233 (Hel.)
Neujean, G., 189 (Rab.), 324 (Typh.)
Neves, A., 1150 (Lep.)
Neveu-Lemaire, M., 1086 (B.R.)
Newcomb, E. H., with Kartman, Campau & Morrison, 716 (Tryp.)
Newman, P. P., (653) (Lep.)
Newton, W. L. & Pratt, I., 52 (Hel.)
——, Wright, W. H. & Pratt, I., 52 (Hel.)
Nichols, E., with Tumulty, Singewald & Lidz, 819 (Mal.)
Nicholls, L., 165 (B.R.), 1062 (Def. Dis.)
Nicolle, P., with Lwoff, (1026) (Tryp.)
Niles, W. J., with Jayewickreme, 1079 (Ent.)
Niño, F., with Taiana & Boragina, 487 (Der.)
Niño, F. L., (227) (Hel.), 588 (Der.)
—— & Fernandez, J. C., (257) (Ent.)
Nisbet, T. W., 481 (Der.)
Nitti, F., Conge, M. & Kauffmann, G., 749 (R.F.)
Niven, J., with van den Ende, Locket, Hargreaves & Lennhoff, 918 (Typh.)
Niven, J. S. F., with Buckland, Dudgeon, Edward, Henderson-Begg, MacCallum, Rowlands & van den Ende, with Bargmann, Curtis & Shepherd, 436 (Typh.)
Nixon, W. C. W., with Eckstein, 623 (Mal.)
Noad, K. B., 1132 (Typh.)
Noble, B. G., 1084 (Reports, etc.)
Nobrega, G., 846 (R.F.)
Noe, W. L., Jr., Greene, C. C., Jr. & Cheney, G., 521 (Mal.)
Nor-el-Din, G., with Halawani, 1109 (Mal.), (1145) (Ys.), (1158) (Hel.)
——, with —— & Awni, 1142 (Am.)
Norman-Walker, J. N., 1089 (B.R.)
Noronha, A. J., (9) (Mal.)
Norris, L. C., with Robertson, Daniel, Farmer & Heuser, 1066 (Haem.)
Notestein, F. W. & Jurkat, E., (261) (Reports, etc.)
Novacovich, G. G., 955 (Der.)
Nuñez Andrade, R., 674 (Der.)
Nyka, W., 119 (Typh.)

O.

Oastler, E. G. & Fidler, H. K., 1029 (Leish.)
Ockuly, E. A., 227 (Hel.)
O'Connor, J. L., 642 (Typh.)
O'Donovan, W. J. & Michaelson, I. C., 1175 (Oph.)
Ogden, F. N., with Zuelzer, 950 (Haem.)
Ogden, L. W., with Rees, 857 (Heat Str.)
Ogilvie, W. H., 251 (Misc. Dis.)
Oldham, F. K. & Kelsey, F. E., 11 (Mal.)
——, with ——, Cantrell & Geiling, (625) (Mal.)
——, with —— & Geiling, 404 (Mal.)
Oleinik, E., with Olitzki, Czaczkes & Kutzenok, (635) (Typh.)
Olitzki, L., Oleinik, E., Czaczkes, J. W. & Kutzenok, A. (635) (Typh.)
Oliveira, E. de S., 940 (Hel.)
Oliver González, J., 357 (Hel.)
——, Biaggi, N. & Rivera León, J., 347 (Hel.)
——, with Culbertson & Rose, 54, 354, 763 (Hel.)
——, with Hernandez Morales, 1156 (Hel.)
——, with —— & Pratt, 1045 (Hel.)
—— & Morales, F. H., 471 (Hel.)
—— & Pratt, C. K., 348 (Hel.)
Oliveros Nava, B., 755 (Hel.)
Olmos Castro, N. & Bonatti, A. A., 849 (Lep.)
——, with ——, 850 (Lep.)
Olson, T. A. & Dahms, R. G., (798) (Ent.)
Omar, M., with Wolman & Abu-Taleb, 338 (R.F.)
Openshaw, H. T., with Hull, Lovell, Payman & Todd, (822) (Mal.)
Orkney, J. M., 501 (Reports, etc.)
Orlov, P. M., with Beklemishev & Shlenova, 1112 (Mal.)
Ortiz, L. F., with Goldman, 664 (Hel.)
Ortiz Marlotte, C., with Bustamante & Varela, 1134 (Typh.)
——, Malo-Juvera, F. & Payne, G. C., 324 (Typh.)
Osborn, S. H., 867 (Reports, etc.)
Osborno-Mesa, E., with Kumm & Boshell-Manrique, 599 (Ent.)
Osgood, S. B., 92 (Mal.)
Osman, A. A., 417 (Bl.)
Osorio, M. T., with Mazzotti, 472 (Hel.)
Ostroumov, V. G., 1142 (Am.)
Oteiza, A. & Leon Blanco, F., 587 (Der.)
Ott, W. H., with Seeler, 412 (Mal.)
Ouary, G., with Moreau, 378 (Ulc.)
Ovalle, H., with Macchiavello, 1033 (Typh.)
——, with —— & Cifuentes, 1032, 1033 (Typh.)
Owen, E. C., (822) (Mal.)
——, with Curd, Davis, Rose & Tuey, (1110) (Mal.)
Owen, W. B., 6 (Mal.)
Owens, B. B., with Curtis, 486 (Der.)
Ozturk, M. H., 157 (Misc. Dis.)

P.

Pacheco-Luna, R., 1159 (Hel.)
Packchanian, A., 723, (1027) (Leish.)
——, with Munter, 721 (Leish.)
Packer, H., 1108 (Mal.)
Paddock, F. K., with Mason, Daniels & Gordon, 1157 (Hel.)
Padilla, E., with Golden, 1185 (Ulc.)
Page, A. B. P., Stringer, A. & Blackith, R. E., 600 (Ent.)
Page, J. A., 1060 (Def. Dis.)
Pal, R., 518 (Mal.)
Palley, A. & Bruwer, T., 1083 (Reports, etc.)
Palmer, E. D., 796 (Ent.)
Pampana, E., 875 (B.R.)

Pang, H. Q., with Lee, 66 (Misc. Dis.)
Pang, K. H., 325 (Typh.)
——, Zia, S. H., Chen, S. M. & Feng, Y. S., 326 (Typh.)
Panja, D., with Ghosh, (490) (Misc.)
Panja, G., 218 (Chl.), 219 (Dys.), 378 (Ulc.), (1127) (Leish.)
—— & Ghosh, S. K., 218 (Chl.)
——, with Gupta & Chatterjee, 1138 (Pl.)
Panthier, R., with Levaditi, 731 (Typh.)
Papafigou, T. (904) (Mal.)
Pará, M., 1169 (Der.)
Paraense, L., 309 (Mal.)
Paraschivescu, N., with Ciuca, Mesrobeanu, Badenski & Munteanu, (210) (Typh.)
Parent, M., with Vincke, 307 (Mal.)
——, with ——, Berteaux & de Meulenaere, 291 (Mal.)
Park, R. G., 64 (Heat Str.)
Parker, R. C. & Hollender, A. J., 186 (Rab.)
Parker, W. V. & Johnson, H. A., 13 (Mal.)
Parkes, J., with Cooke, Elkes, Frazer & Peeney, 946 (Sp.)
Parrot, G., 328 (Typh.)
Parrot, L. & Malbrant, R., (160) (Ent.)
—— & Martin, R., (601) (Ent.)
——, Mornet, J. & Cadenat, J., (160), (602) (Ent.)
——, with Sergent & Donatien, (603) (Ent.)
Parry, E., 624 (Mal.)
Parry, T. E., with Tattersall, 430 (Typh.)
Pascale, H., 969 (Reports, etc.)
—— & Cruz, E., 546 (Typh.)
Paseyro, P., (376) (Haem.)
Patel, J. C., 220, 221 (Am.)
Paul, B. M., with Gupta, Chatterjee & Ghose, 130 (Chl.)
de Paula Souza, G. H., 126, 1135 (Y.F.)
Pavillard, S. S., 489 (Misc. Dis.)
Pavlovskiĭ, E. N. & Skruinnik, A. N., 223 (R.F.)
Pavlovsky, E. N., 65 (Misc. Dis.), 743 (R.F.)
—— & Kuzmina, L. A., 744 (R.F.)
Payman, L. C., with Hull, Lovell, Openshaw & Todd, (822) (Mal.)
Payne, G. C., with Ortiz-Mariotte & Malo-Juvera, 324 (Typh.)
Pecoraro, V., with Cerboni & Rodolfo Mercau, 847 (Lep.)
Peddie, J. J. G., with Cottrell, 333 (Am.)
Peel, E. & Chardome, M., 1159 (Hel.)
——, with van Hoof, Lewillon, Henrard & Rodjestvensky, 536 (Tryp.)
——, with Wanson & Henrard, 233 (Hel.)
Peeney, A. L. P., with Cooke, Elkes, Frazer & Parkes, 946 (Sp.)
Pepper, D. S., with Elson & Forrester, 556 (Dys.)
Pepper, O. H. P. & Díaz Rivera, R. S., 356 (Hel.)
Pereira, A. C., 1151 (Lep.)
Pereira, O. de L., 609 (B.R.)
Pereira, P. C. R., 564 (Lep.)
Pérez, A. & Romaña, C., 112 (Tryp.)
Pérez Ara, A. & Pérez Vigueras, I., 580 (Hel.)
Pérez Vigueras, I., with Pérez Ara, 580 (Hel.)
Perret-Gentil, A., 96, 200 (Mal.)
Perrin, T. L., with Ashburn, Brady & Lawton, 662 (Hel.)
Perry, D. J., with Spies, Cogswell & Frommeyer, 60 (Def. Dis.)
Perves, M., (524) (Mal.)
Peset Aleixandre, T. & Romeo Viamonte, J. M., (196) (Mal.)
Pessôa, S. B. & Amaral, D. F., 43 (Am.)
—— & Barretto, M. P., 1030, 1127, 1128 (Leish.)
Peters, C. N., Huntress, R. L. & Porter, J. E., (346) (Hel.)
Peters, J. T., 246 (Der.), 534 (Bl.)
Pfanner, E. F., 674 (Der.)
Philip, C. B., 554 (Den.)
—— & Kohls, G. M., 119 (Typh.)
——, Woodward, T. E. & Sullivan, R. R., 733 (Typh.)
——, with —— & Loranger, 836 (Typh.)
Philip, M. I., Ramakrishna, V. & Rao, V. V., 203 (Mal.)
Philipzs, G. C. L., with Marray, (136) (Am.)
Picard, H. & Bensimon, T., 42 (Am.)
Piers, F., (1084) (Reports, etc.)
Pierson, J., with Seeler & Malanga, 309 (Mal.)
Pike, J. B., with Taft, 447 (R.F.)
Piney, A., & Hamilton-Paterson, J. L., 870 (B.R.)
Pinkerton, H., with Greiff, 22 (Typh.)
——, with Settle & Corbett, 27 (Typh.)
Pinto, C., 163 (Reports, etc.)
——, with Torres, 658, 1156 (Hel.)
Pinto, H., (1027) (Leish.)
Pinto, M. R., with de Azevedo & Cambournac, (716), 828, (829) (Tryp.)
Pippard, J. S., with Hutchison & Gleeson-White, 338 (R.F.)
Pirot, R. & Bourgain, M., 336, 337 *bis* (R.F.)
Pittar, C. A., 1176 (Oph.)
Pittendrigh, C. S., with Downs, 1001 (Mal.)
Planchart, M. A., with Sanabria, 156 (Misc. Dis.)
Platt, B. S., 362 (Def. Dis.)
Plaut, A., 1190 (Prot.)
Plessen, M., (853) (Hel.)
Plötner, K., 393 (Mal.)
Plotz, H., with Bell, 434 (Typh.)
——, Bennett, B. L. & Reagan, R. L., 920 (Typh.)
—— & Wertman, K., 429 (Typh.)
——, —— & Bennett, B. L., 725 (Typh.)
Podlesker, A., 13 (Mal.)
Pogge, R. C., 456 (Lep.)
——, with Faget, 343, 460 (Lep.)
Polak, M. F., 264 (B.R.)
Pollard, M., with Davis, 1033 (Typh.)
——, Livesay, H. R., Wilson, D. J. & Woodland, J. C., 738, 925 (Den.)
Polson, A., Joubert, F. J. & Haig, D. A., (954) (Vms.)
Polumordvinov, A. D., 283 (Mal.)
Pondé, A., with Barreto, (424) (Tryp.)
Ponder, E., 61 (Haem.)
Porter, J. E., with Peters & Huntress, (346) (Hel.)
Porto, A., with Rocha e Silva & Andrade, 1052 (Hel.)
Portugal, H. & Rocha, G. L., 1148 (Lep.)
Possolo, H., 266 (B.R.)
Post-Graduate Med. J., (939) (Hel.)
Potter, C. & Gillham, E. M., 1193 (Ent.)

Poursines, Y., with Cornil & Moustardier, 216 (Pl.)
Power, S., 677 (Misc. Dis.)
Powning, R. F., 384 (Ent.)
Pozzo, A., 880 (B.R.)
Prabhu, M. N., 653 (Lep.)
Pratt, C. K., 470 (Hel.)
——, with Hernandez-Morales & Oliver-González, 1045 (Hel.)
——, with Oliver González, 348 (Hel.)
Pratt, I., with Newton, 52 (Hel.)
——, with —— & Wright, 52 (Hel.)
Prendiville, J. T., with Lehmann, 787 (Prot.)
Preuss, with v. Bormann, 324 (Typh.)
Price, M. M. & Lyman, F. E., 14 (Mal.)
Proc. Roy. Soc. Med., 835 (Typh.), 944 (Def. Dis.), 1040 (Am.)
Prokopenko, L. I., 8, 300, 701 (Mal.)
Prost, P., with Farinaud, (285) (Mal.)
Prout, C., with Heidelberger, Hindle & Rose, 1013 (Mal.)
Prudhomme, R. O., 657 (Lep.)
Puig Solanes, M., Fontes, A. & Antonio Quiroz, J., 764 (Hel.)
Pullar, E. M., (1048) (Hel.)
Pullen, R. L., with Stuart, 836 (Typh.)
Pullman, T. N., with Heidelberger, Mayer, Alving, Craige, Jones & Whorton, 1013 (Mal.)
Puntoni, V., 264 (B.R.)
Putnam, P., with Russell & Rao, 196 (Mal.)
Pyles, W. J., with Gellhorn, van Dyke & Tupikova, 722 (Leish.)

Q.

Queen, F. B., with Sweeney & Friedlander, 114 (Leish.)
Quitter, F., 144 (Def. Dis.)

R.

Raboni, C., (705) (Mal.)
Radford, C. D., 683 *bis* (Ent.)
Radna, R., 1041 (Am.), 1146 (Lep.)
Raettig, H., 124, 211 (Typh.)
Raffaele, G., 1016 (Mal.)
Rafyi, A., 1144 (R.F.)
Rai Chaudhuri, M. N., with Chaudhuri, 843, (844) (Am.)
Raison, C. G., (822) (Mal.)
Rakshit, P. C., with Bose & Ghosh, 928 (Dys.)
Ramachandran, S., with Krishnan & Sadhu, 473 (Def. Dis.)
Ramakrishna, V., with Philip & Rao, 203 (Mal.)
Ramos, J., Jr. & Laus F., J. A., 909 (Tryp.)
Randolf, N. M., (1123) (Tryp.), 1145 (R.F.)
Rangam, C. M., with Reddy, 444 (Am.)
Rao, S. S., 231 (Hel.)
Rao, T. R., 519 (Mal.)
——, with Russell & Putnam, 196 (Mal.)
Rao, V. G., 1075 (Misc. Dis.)
Rao, V. V., with Philip & Ramakrishna, 203 (Mal.)
Raper, A. B., Wilson, M. E. & Wilson, D. B., 8 (Mal.)
Raphael, M., with Browning, Klein & Coblenz, 548 (Typh.)
Rappaport, F. & Eichhorn, F., (1164) (Haem.)
Rapport, M. M., with Mead, Senear, Maynard & Koepfli, 823 (Mal.)
Ratcliffe, A. W., 967 (Lab.)
Ray, J. C., with Ghosh, H., & Ghosh, N. N., 1125 (Leish.)
Re, P. M., with Jorge, 1048 (Hel.)
Read, H. S., with Kaplan & Becker, 1110 (Mal.)
Reagan, R. L., with Plotz & Bennett, 920 (Typh.)
Reardon, L. V., with Rees, 556 (Am.)
Reddy, D. G. & Rangam, C. M., 444 (Am.)
—— & Thangavelu, M., 495 (Lab.)
Rees, C. W. & Reardon, L. V., 556 (Am.)
Rees, W. H. & Ogden, L. W., 857 (Heat Str.)
Rein, C. R., with Cooper & Beard, 1027 (Leish.)
——, with Dwindelle, Sternberg & Sheldon, 1043 (Ys.)
Reingold, I. M. & Webb, F. R., 581 (Def. Dis.)
Reinhart, J. B., with Harrell, Wolff & Venning, 923 (Typh.)
Remlinger, P., (196) (Rab.)
Renbourn, E. T., 857 (Heat Str.)
Rengifo, S., with Gast Galvis, 113 (Leish.)
Renshaw, R. J. F., with Kiskaddon, 158 (Prot.)
Reppert, L. B., with Wright & Cutting, 781 (Heat Str.)
Resseler, R., with Rodhain, 424 (Tryp.)
Rev. Paludisme et Méd. Trop., (307) (Mal.)
Revista Brasileira de Leprologia, 451 (Lep.)
Rey, H., with Huffaker & Soto, 392 (Mal.)
——, Soto, H. & Huffaker, C. B., (816) (Mal.)
Rhodesia, Southern, 1201 (Reports, etc.)
Ribbands, C. R., 409, 621, 904, 1014 (Mal.)
Ricci, M., (852) (Hel.)
Rich, W. M., with Dansey-Browning, 588 (Oph.)
Richardson, A. P., Walker, H. A., Loeb, P. & Miller, I., 446 (R.F.)
Richardson, D. N., (822) (Mal.)
Richardson, D. T., 690 (B.R.)
Richet, C., (116) (Typh.)
Richter, C. P., 331 (Pl.)
Ridley, H., 55 (Hel.), 534, 1018 (Tryp.), 588 (Oph.)
Riemerschmid, G., 248 (Heat Str.)
Rifkin, H., Cekada, E. B., Zarrow, M., Henderson, D. G. & Whitehead, J. O., 188 (Rab.)
—— & Thompson, K. J., 353 (Hel.)
——, with —— & Zarrow, 468 (Hel.)
——, with Zarrow, 571 (Hel.)
Rigby, D. J., with Maegraith, Adams, King, Tottey & Sladden, 821 (Mal.)
Rigdon, R. H., 715, 1119 (Mal.)
——, with Marvin, 715 (Mal.)
——, with Rostorfer, 309 (Mal.)
—— & Rudisell, H., Jr., 310 (Mal.)
—— & Varnadoe, N. B., 630 (Mal.)
Rights, F. L., with Smadel & Jackson, 547, 919 (Typh.)
Rigler, R., 116 (Typh.)
Riley, R. L., with Goodhue, 962 (Ent.)
Riseman, J. E. F., with Freedberg, 245 Vms.)

Rivera León, J., with Oliver González & Biaggi, 347 (Hel.)
Robertson, E. I., Daniel, L. J., Farmer, F. A., Norris, L. C. & Heuser, G. F., (1066) (Haem.)
Robinson, G., 243 (Haem.)
Robinson, H. M., Jr. & McKinney, W. W., 298 (Mal.)
Roblin, R. O., Jr., with English, Clark, Shepherd, Marson & Krapcho, (905) (Mal.)
Roca-Garcia, M., with Bates, 32, 328 (Y.F.)
Rocha, G. L., with Portugal, 1148 (Lep.)
Rocha e Silva, M. & Graña, R., 1052 *bis* (Hel.)
——, Porto, A. & Andrade, S. O., 1052 (Hel.)
Rodhain, J., (908) (Tryp.)
—— & Resseler, R., 424 (Tryp.)
Rodjestvensky, B., with van Hoof, Lewillon, Henrard & Peel, 536 (Tryp.)
Rodolfo Mercau, A., with Cerboni & Pecoraro, 847 (Lep.)
Rodriguez, H., H., 188 (Rab.)
Rodriguez, J. N., with Bancroft, Guinto & Marques, 454 (Lep.)
——, with Doull, Guinto & Bancroft, 453 (Lep.)
Rodriguez-Molina, R., 239, (241) (Sp.)
Rodriguez Pascual, C., (935) (Lep.)
Rogan, J. M. & Coombes, A. E. R., 524 (Mal.)
Roger, H., (229) (Hel.)
Rogers, A. M. & Dammin, G. J., 1049 (Hel.)
Rogers, E. W., with Marshall, 98 (Mal.)
Rogers, K. B., 392 (Mal.), 1081 (Lab.)
Rogers, L., 1153 (Lep.)
—— & Muir, E., 689 (B.R.)
Romaña, C., 111 (Tryp.)
——, with Larcher, 110 (Tryp.)
——, with Pérez, 112 (Tryp.)
—— & Terracini, E., 110, (110) (Tryp.)
Romeiro, O. dos S., (1123) (Tryp.)
Romeo, B. J., 1133 (Typh.)
Romeo Viamonte, J. M., 817 (Mal.)
—— & Irigoyen Ramirez, A., (197) (Mal.)
——, with Peset Aleixandre, (196) (Mal.)
Romero, H., with Viel, 545 (Typh.)
Romijn, C., 1164 (Haem.)
Rorich, P. R., with Cluver & Jokl, (1084) (Reports, etc.)
Rose, A. S., with Heidelberger, Prout & Hindle, 1013 (Mal.)
Rose, F. L., with Basford & Curd, (1110) (Mal.)
——, with Curd, (822 *bis*), (1110) (Mal.)
——, with ——, Davis, Owen & Tuey, (1110) (Mal.)
——, with —— & Davey, 394, 400 (Mal.)
Rose, H. M., Culbertson, J. T. & Lipman, M.O., 232 (Hel.)
——, with —— & Oliver-Gonzalez, 54, 354, 763 (Hel.)
——, Duane, R. B. & Fischel, E. E., 433 (Typh.)
Rosenbaum, S., 147 (Haem.)
Rosenthal, J., 1069 (Der.)
Rostorfer, H. H. & Rigdon, R. H., 309 (Mal.)
Rotberg, A., with Bechelli & Keil, 225 (Lep.)
Roth, H., 359 (Hel.)
Rotman, C. M. H., 34 (Pl.)
Roubaud, E., (290) (Mal.), 419 (Tryp.)
—— & Caubet, P., 418 (Tryp.)
—— & Colas-Belcour, J., 383 (Ent.)
——, Stefanopoulo, G. J. & Duvolon, S., Mlle., 419 (Tryp.)
Rouvier, J., with Despujols, Bergeret & Calmet, (253) (Misc. Dis.)
Row, R., 1147 (Lep.)
Rowe, H. P., 79 (Reports, etc.)
Rowlands, J. S. F., with Buckland, Dudgeon, Edward, Henderson-Begg, MacCallum, Niven, Rowlands & van den Ende, with Bargmann, Curtis & Shepherd, 436 (Typh.)
Roy, B. C., 725 (Typh.)
Roy, D. N., 871 (B.R.)
Roy. Nav. Med. Bull., 244 (Vms.)
Rozeboom, L. E., with Belkin & Knight, 6 (Mal.)
—— & Knight, K. L., 817 (Mal.)
Rubenstein, A. D., Shulman, M. H. & Merrill, D., 292 (Mal.)
Rubin, S. S., (543) (Typh.)
Rudisell, H., Jr., with Rigdon, 310 (Mal.)
Rudra, M. N. & Bhattacharya, K. P., 787 (Misc. Dis.)
Ruegamer, W. R., with Cooperman & Elvehjem, (1067) (Haem.)
——, with ——, —— & McCall, 772 (Haem.)
Rugiero, H. R. & Cohen, J., 326 (Typh.)
Ruiz Cestero, G., with Hernández Morales, (228) (Hel.)
Rumreich, A. S. & Koepke, J. A., 429 (Typh.)
—— & Wynn, R. S., (35) (Pl.)
Ruppol, A., 202 (Mal.)
Rushcliffe, 85 (B. R.)
Russell, J. C., with Eyles & Sabrosky, 199 (Mal.)
Russell, P. F., (620) (Mal.)
——, Rao, T. R. & Putnam, P., 196 (Mal.)

S.

de Sá, J. M., with Gama, 936 (Hel.)
Sabrosky, C. W., with Eyles & Russell, 199 (Mal.)
Sachs, A., 729 (Typh.)
Sadhu, K., with Krishnan & Ramachandran, 473 (Def. Dis.)
Sadler, G. G., with Kitchen, 415 (Bl.)
Sadusk, J. F., Jr., with Blake, Maxcy, Kohls & Bell, 327 (Typh.)
Sagatelova, I. S., with Kalandadze, 1106 (Mal.)
Sagher, F., 1028 (Leish.)
——, with Dostrovsky, 1030 (Leish.)
Salgado, P., with Convit, Azulay & Bermudez, 457 (Lep.)
Sanabria, A. & Planchart M., A., 156 (Misc. Dis.)
Sanchez Garcia, E., with Leon y Blanco, 586 (Der.)
Sanderson, G., with Adams, 396 *quat.* (Mal.)
Sandground, J. H., with Shrapnel & Johnson, 928 (Am.)
Sandler, A., 1037 (Den.)
Sangster, C. B., 759 (Hel.)
—— & Kay, H. B., 29 (Typh.)
Sanyal, S. C. (1190) (Misc. Dis.)

Saphir, W., 54 (Hel.), 377 (Heat Str.)
Sardana, M. N., 1182 (Oph.)
Saslaw, S., with Wilson & Doan, (952) (Haem.)
Sasportas, L., 282 (Mal.)
Satti, M. H., with Horgan, 17 (Leish.)
Sautet, J. & Audibert, Y., 789 (Ent.)
—— & Marneffe, H., (258) (Reports, etc.), 462 (Hel.)
Savino, E., 36 (Pl.)
Saxton, W. J., Hatcher, F. & Derrick, E. H., 1169 (Der.)
Sayers, M. H. P., 1132 (Typh.)
Scadding, J. G., 132 (Dys.)
Schaafsma, A., with Grasset & Hodgson, 670 (Vms.)
Schäfer, K. H., 210 (Typh.)
Schallek, W., with Welsh, (1080) (Ent.)
Scharff, J. W., 203 (Mal.)
——, with White, S., 203 (Mal.)
Schatz, A., Magnuson, H. J., Waksman, S. A. & Eagle, H., 1025 (Tryp.)
Scheie, H. G., 1178 (Oph.)
Scheinker, I. M., with Blankenhorn, Vilter & Austin, 945 (Def. Dis.)
Schenkein, E. L., with Freund, Thomson, Sommer & Walter, 311 (Mal.)
Schieber, C., 374 (Harm.)
Schlosser, R. J., 575 (Hel.)
Schmidt, W., 117 (Typh.)
Schmitt, C. L., Alpins, O. & Chambers, G., 482 (Der.)
Schneider, M., 1170 (Oph.)
Schneyder, R., with Liger, (939) (Hel.)
Schoetter, M., with Devignat & Gille-Simul, 330 (Pl.)
Schofield, A. L., with Wolfe, 762 (Hel.)
Schofield, K., with Simpson, (625) (Mal.)
Schönbrunner, E., 118 (Typh.)
Schorr, S., with Druckman, 133 (Am.)
Schroeder, E. F., with Most, London, Kane, Lavietes & Hayman, 1011 (Mal.)
Schroeder, H. O. & Lindquist, A. W., 963 (Ent.)
——, with ——, Travis, Madden & Jones, 159 (Ent.)
——, with ——, Wilson & Madden, 159 (Ent.)
Schüffner, W., 233 (Hel.)
Schüffner, W. A. P., with Swellengrebel, (355) (Hel.)
Schuhardt, V. T. & Hemphill, E. C., 748 (R.F.)
Schujman, S., 846, 1152 (Lep.)
Schultes, W., 199 (Mal.)
Scott, H., 317 (Leish.)
Scott, H. H., 690 (B.R.)
Scott, J. A., 465, 1050 (Hel.), (969) (Reports, etc.)
Seal, S. C., 217 (Chl.)
——, with Sen, 580 (Def. Dis.)
Sebrell, W. H., with Hundley, 1061 (Def. Dis.)
——, with King, 854 (Def. Dis.)
Seeler, A. O., Malanga, C. & Pierson, J., 309 (Mal.)
—— & Ott, W. H., 412 (Mal.)
Seheult, R., (67) (Misc. Dis.)
Seite, P., with Le Gac & Combescot de Marsaguet, 621 (Mal.)
Sekar, C. C., with Dutta & Mukerji, 1029 (Leish.)
Selzer, I., with Sharnoff & Geiger, 1002 (Mal.)
Sen, A. K., 953 (Haem.)
Sen, K. & Ghose, N., 576 (Hel.)
Sen, P. C., with Seal, S. C., 580 (Def. Dis.)
Sen, R. L., with Das Gupta, 1145 (R.F.)
Sen Gupta, K. K., with Mukerji, 230 (Hel.)
Sen Gupta, P. C., 425, 722 (Leish.)
——, with Bose & Basu, 219 (Dys.)
—— & Chakravarty, N. K., 833, 834 (Leish.)
Senear, A. E., with Mead, Rapport, Maynard & Koepfli, 823 (Mal.)
Senekjie, H. A. & Lewis, R. A., 320 (Leish.), 677 (Prot.)
Sergent, E., 560 (R.F.)
——, Donatien, A. & Parrot, L., (603) (Ent.)
Sergent, Et., 148 (Vms.)
Seshadrinathan, N., 251 (Ulc.)
Settle, E. B., Pinkerton, H. & Corbett, A. J., 27 (Typh.)
Sezonismo, Lisbon, 903 *sex* (Mal.)
Shah, M. A., 1175 (Oph.)
Shallenberger, P. L., with Ginsberg, 1007 (Mal.)
Shapiro, S., with Murphy, 581 (Haem.)
Sharnoff, J. G., Geiger, J. & Selzer, I., 1002 (Mal.)
Shear, M. J., with Topping & Bengtson, 18 (Typh.)
Sheard, C., with Kierland, Mason & Lobitz, 1008 (Mal.)
Sheehan, H. L., 338 (R.F.)
Sheldon, A. J., with Dwindelle, Rein & Sternberg, 1043 (Ys.)
Shelley, W. B. & Horvath, S. M., 675 (Heat Str.)
Shepard, C. C., 21 (Typh.)
——, with Irons, Topping & Cox, 924 (Typh.)
—— & Topping, N. H., 21, 735 (Typh.)
——, with ——, 910, 911 (Typh.)
—— & Wyckoff, R. W. G., 913 (Typh.)
Shepherd, M. A., with Buckland, Dudgeon, Edward, Henderson-Begg, MacCullum, Niven, Rowlandy, van den Ende, Bargmann & Curtis, 436 (Typh.)
Shepherd, M. M., 782 (Ulc.)
Shepherd, R. G., with English, Clark, Marson, Krapcho & Roblin, (905) (Mal.)
Sherif, M. A. F., with Amin & Mahfouz, 43 (Am.)
Shields, J. B., Johnson, B. C., Hamilton, T. S. & Mitchell, H. H., 591 (Heat Str.)
Shiers, D., 706 (Mal.)
Shih Lu Chang, 37, 222 (Am.)
Shivers, J. C., with Breslow, Walker, Yost & Hauser, (406) (Mal.)
Shlenova, M. F., with Beklemishev & Orlov, 1112 (Mal.)
Shlimovitz, N., 750 (Lep.)
——, with Wartman, 1003 (Mal.)
Shortt, H. E., 317 (Leish.), 684 (Reports, etc.), 1132 (Typh.)
Shoukri, Y. S., 116 (Typh.)
Shourie, K. L., 492 (Misc.)
Shrapnel, B. C., Johnson, C. M. & Sandground J. H., 928 (Am.)
Shrivastava, D. L., with Bhatnagar, 840 (Pl.)

Shropshear, G., with Vaughn & Howser, (157) (Misc. Dis.)
Shulman, M. H., with Rubenstein & Merrill, 292 (Mal.)
Shumeiko, A. I., 48 (Hel.)
Shute, P. G., 8, (393) (Mal.)
Sicé, A., 257 (Reports, etc.)
Siddons, L. B., with Das Gupta, 4 (Mal.)
Silber, R. H., with Edison & Tennent, 363 (Def. Dis.)
Silva, R. & Kopciowska, 428 (Typh.)
da Silva, E. M., 668 (Haem.)
da Silva, P. B., 1158 (Hel.)
da Silva-Lacaz, C., (151) (Der.)
de Silva, S., 40, 443 (Am.)
Silverman, D. N. & Leslie, A., 39 (Am.)
Simmons, E. L., with Taliaferro & Taliaferro, 629 (Mal.)
Simmons, J. S., Whayne, T. F., Anderson, G. W., Horack, H. M. & collaborators, 86 (B.R.)
Simmons, S. W., *et al.*, 104 (Mal.), 789 (Ent.)
——, with Fay & Clapp, 790 (Ent.)
——, with Stierli & Tarzwell, 792 (Ent.)
——, with Upholt, Gaines & Arnold, 794 (Ent.)
Simões, J. M. P., with Cambournac, 95, 306 (Mal.)
Simonin, P. & Girard, J., (478) (Haem.)
Simonsen, D. G., with Moore, Kessel, Marmorston, Llewellyn, Kaplan, Golden, Anderson & Jaffe, (650) (Dys.)
Simpson, J., 944 (Def. Dis.)
Simpson, J. C. E. & Schofield, K., (625) (Mal.)
Singewald, M. L., with Tumulty, Nichols & Lidz, 819 (Mal.)
Singh, B., 238 (Sp.)
Singh, I. & Singh, I., (633) (Bl.)
——, with Singh, (633) (Bl.)
Skruinnik, A. N., with Pavlovskiĭ, 223 (R.F.)
Sladden, R. A., with Maegraith, Adams, King, Tottey & Rigby, 821 (Mal.)
Sloan, N. R., 460, (1153) (Lep.)
Sloan, T. B. M., 655 (Lep.)
Smadel, J. E., Rights, F. L. & Jackson, E. B., 547, 919 (Typh.)
Smart, G. A. & Daley, R., 1160 (Sp.)
Smart, J., 494 (Ent.)
Smith, A. & Greaves, D. P., 965 (Ent.)
Smith, C. S., with Knowles, 710 (Mal.)
Smith, C. N. & Gouck, H. K., (494), 1198 (Ent.)
Smith, D. T., with Weeks, 654 (Lep.)
Smith, F., with Donovick & Farrell, 635 (Typh.)
Smith, G. E., with Metcalf, Hess, Jeffery & Ludwig, 102 (Mal.)
Smith, H. M., (954) (Vms.)
Smith, L. A., 440 (Dys.)
Smith, L. H. & Stoeckle, J. D., 1109 (Mal.)
Smith, P. K., 1131 (Typh.)
Smith, R. E., Stormont, R. T., Bianco, A. A. & Evans, R. L., (1157) (Hel.)
Smith, R. L., 281 (Mal.)
Smithburn, K. C., 214 (Y.F.), 859 (Misc. Dis.)
—— & Mahaffy, A. F., 34 (Y.F.)
Smyth, M. J., 1140 (Am.)
Sneddon, I. B., with Clarke, 944, 1059 (Def. Dis.)
Snell, A. M., 1039 (Am.)
Snyder, F. M. & Morton, F. A., 1196 (Ent.)
Snyder, J. C., with Ecke, Gilliam, Yeomans, Zarafonetis & Murray, 545 (Typh.)
——, with Murray & Zarafonetis, 433 (Typh.)
——, with Yeomans & Gilliam, 24 (Typh.)
——, with ——, Murray, Ecke & Zarafonetis, 636 (Typh.)
—— & Zarafonetis, C. J. D., 433 (Typh.)
——, with ——, Ecke, Yeomans & Murray, 912 (Typh.)
——, —— & Liu, W. T., 426 (Typh.)
——, with —— & Murray, 922 (Typh.)
Sobrinho, O. P., 775 (Vms.)
——, with Versiani & Martins, 754 (Hel.)
Sodeman, W. A. & Lewis, B. O., 135 (Am.)
Sofia, F. & Ciaravino, E., 37 (Am.)
Sofiev, M. S. & Leonova, N. A., 744 (R.F.)
Sokoloff, B., 557 (Am.)
Sokolow, M. & Garland, L. H., 432 (Typh.)
Sollmann, T., with Luckiesh, Taylor & Cole, 675 (Heat Str.)
Soman, D. W. & Nail, S. K., 439 (Chl.)
Somers, R. B. U., 327 (Typh.)
Somervell, T. H., 1040 (Am.)
Sommer, H. E., with Freund, Thomson, Walter & Schenkein, 311 (Mal.)
——, with —— & Walter, 311 (Mal.)
Sompayrac, L. & Spence, H., (1145) (Ys.)
Sonneborn, D. G., 583 (Vms.)
Soto, C. M., with Fernández, 850 (Lep.)
Soto, H., 815 (Mal.)
——, with Huffakerj & Rey, 392 (Mal.)
——, with Rey & Huffaker, (816) (Mal.)
Sotolonga, F., Alfonso, J. & Del Valle-Alemàn, S., (50) (Hel.)
Soule, M. H., with Lofgren, 335, (446) (R.F.)
South Africa, Union of, 999 (Mal.)
de Souza-Araujo, H. C., 655 (Lep.)
South African Med. J., 498 *ter* (Reports, etc).
South African Red Cross Society (Natal Branch), 665 (Def. Dis.)
Souza, P. R., with Campos, 47 (Lep.)
Spadaro, O., 41 (Am.)
Sparkman, R. E., with Kiker, 308 (Mal.)
Speck, J. F., Moulder, J. W. & Evans, E. A., Jr., 1116 (Mal.)
Spector, H. & Hamilton, T. S., (378) (Heat Str.)
——, —— & Mitchell, H. H., 591 (Heat Str.)
——, Mitchell, H. H. & Hamilton, T. S., (378), 591 (Heat Str.)
Spector, S., Haviland, J. W. & Coggeshall, L. T., 823 (Mal.)
Spence, H., with Sompayrac, (1145) (Ys.)
Spencer, G. J., 865 (Lab.)
Spicer, S. S., 282 (Mal.)
Spies, T. D., 582, 771 (Haem.)
——, Frommeyer, W. B., Garcia Lopez, G., Lopez Toca, R. & Gwinner, G., 949 (Sp.)
——, Lopez, G. G., Menendez, J. A., Minnich, V. & Koch, M. B., 474 (Sp.)
——, Milanes, F., Menéndez, A., Koch, M. B. & Minnich, V., 769 (Sp.)
—— & Morey, M. M., 768 (Def. Dis.)
——, Perry, D. J., Cogswell, R. C. & Frommeyer, W. B., 60 (Def. Dis.)
——, Vilter, C. F., Cline, J. K. & Frommeyer, W. B., 772 (Haem.)

Spies, T. D. with Vilter, C. F., & Koch, 375 (Haem.)
——, ——, —— & Caldwell, M. H., 375 (Haem.)
Spillane, J. D., 944 (Def. Dis.)
Spinks, A., 398 (Mal.)
—— & Tottey, M. M., 398 *bis*, 400, 707 (Mal.)
——, —— & Maegraith, B. G., (707) (Mal.)
Spitz, S., with Allen, 25 (Typh.)
——, with Ash, 691 (B.R.)
Sprecher, A., 849 (Lep.)
Springer, L., with Carini, (1170) (Der.)
Stager, K. E., with Mackie, Davis, Fuller, Knapp, Steinacker, Traub, Jellison, Millspaugh, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Stammers, F. M. G. & Whitfield, F. G. S., 865 (Ent.)
Stanbury, J. B. & Huyck, J. H., (257) (Ent.)
Stannus, H. S., 684 (Reports, etc.)
Stare, F. J., with Williamson, Hegsted & McKibbin, 1009 (Mal.)
Stefanopoulo, G., Caubet, P. & Duvolon, S., Mlle., 417, 420 (Tryp.)
——, with Lavier, 463 (Hel.)
Stefanopoulo, G. J., with Roubaud & Duvolon, Mlle., 419 (Tryp.)
Steinacker, M. L., with Mackie, Davis, Fuller, Knapp, Stager, Traub, Jellison, Millspaugh, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Steinle, M. L., with Lackey, 794 (Ent.)
Stella, E., (864) (Ent.)
Stelma, T., with Wharton, 1054 (Hel.)
Stenning, J. C., 488 (Heat Str.)
Stephan, E., (1077) (Misc. Dis.)
Sternberg, T. H., with Dwindelle, Rein & Sheldon, 1043 (Ys.)
Stevens, T. S., with Fulton, 1121 (Tryp.)
Stevenson, D. S., 407 (Mal.)
Stevenson, I. P., (1164) (Haem.)
Stewart, J. L., 907 (Tryp.)
Stewart, M. O., with Florio, Hammon & Laurent, 737 (Den.)
——, with —— & Mugrage, 438 (Den.)
Steyn, D. G., with van der Walt, 960 (Misc. Dis.)
Stierli, H., Simmons, S. W. & Tarzwell, C. M., 792 (Ent.)
——, with Tarzwell, 791 (Ent.)
Stifler, W. C., Jr., with Brown & Bethea, 939 (Hel.)
Stock, F. E., 1171 (Oph.)
Stock, P. G., 124 (Y.F.)
Stockdale, F., 70 (Reports, etc.)
Stoeckle, F. D., with Smith, 1109 (Mal.)
Stokes, J. F., 299 (Mal.)
Stone, A., 6 (Mal.)
Stonham, F. V., 705 (Mal.)
Stormont, R. T., with Smith, Bianco & Evans, (1157) (Hel.)
Stowman, K., 128 (Chl.)
Stratman-Thomas, W. K., with Knisely, Eliot & Bloch, 1113 (Mal.)
Stringer, A., with Page & Blackith, 600 (Ent.)
Stuart, B. M. & Pullen, R. L., 836 (Typh.)
Stuart, G., 44 (R.F.)
Stubbs, R. E., with Moyne, Crowdy, Citrine, Mackinnon, Blacklock, Engledow & Henderson, 70 (Reports, etc.)
Stubbs, T. H., with Boyd & Weinstein, 1200 (Reports, etc.)
——, with Moore, Young & Hardman, 1002 (Mal.)
——, with Young, Moore, Ehrman, Hardman, Ellis & Burgess, 5 (Mal.)
Stubenbord, W. D., 1045 (Ys.)
—— & Allen, R. F., 1050 (Hel.)
Stühmer, A., 123 (Typh.)
Suárez, R. M. Díaz Rivera, R. S. & Hernández Morales, F., 496 (Lab.)
—— & Hernández Morales, F., 349 (Hel.)
Subbarow, Y., with Little, 538 (Tryp.)
Subramaniam, R., 457 (Lep.), (852) (Hel.)
Suessenguth, H. & Kline, B. S., 360 (Hel.)
Sullivan, R. R., with Philip & Woodward, 733 (Typh.)
Sullivan, T. de S., with Davis, 1026 (Tryp.)
Sulzberger, M. B., Addenbrooke, E. F., Joyce, S. J., Greenberg, S. & Mack, A. G., 1166 (Der.)
Summers, W. A., with Harris, 569 (Hel.)
Surdey, with Watrin, (487) (Der.)
Sureau, B., 323 (Typh.)
——, with Giroud, (324) (Typh.)
——, with Martin, Le Roy, Babouot & Bourcart, 464 (Hel.)
Susman, E., with Fay, 781 (Heat Str.)
Sutliff, W. D. & Antonio Echandi, G., R., (1057) (Hel.)
Swaroop, S., with Yacob, 74 (Reports, etc.), 285 (Mal.)
Swarts, W. B. & Wanamaker, J. F., 966 (Ent.)
Sweeney, J. S., Friedlander, R. D. & Queen, F. B., 114 (Leish.)
Sweetman, H. L., 1194, (1195) (Ent.)
Swellengrebel, N. H. & Schüffner, W. A. P., (355) (Hel.)
——, with van der Kuyp, E., 267 (B.R.)
Sylla, A., 114 (Typh.)

T.

Taft, W. C. & Pike, J. B., 447 (R.F.)
Taiana, J. A., Boragina, R. C. & Niño, F., 487 (Der.)
Taliaferro, L. G., with Taliaferro & Simmons, 629 (Mal.)
Taliaferro, W. H., 414 (Mal.)
—— & Bloom, W., 630 (Mal.)
——, Taliaferro, L. G. & Simmons, E. L., 629 (Mal.)
Talice, R., 692 (B.R.)
Talice, R. V., 112 (Tryp.)
—— & López-Fernández, J., 112 (Tryp.)
—— & Mackinnon, J. E., 292 (Mal.)
Tan, C. C. & Liu, Y., 333 (Am.)
Tang, I. L., with Chen & Wang, (97) (Mal.)
Tanganyika Territory, 79 (Reports, etc.)
Tarnoky, A. L., with King & Gilchrist, (707) (Mal.)
Tarr, L., 657 (Hel.)
Tarzwell, C. M. & Stierli, H., 791 (Ent.)
——, with —— & Simmons, 792 (Ent.)

Tattersall, R. N., 28 (Typh.)
—— & Parry, T. E., 430 (Typh.)
Taylor, A. H., with Luckiesh, Cole & Sollmann, 675 (Heat Str.)
Taylor, C. E., 342 (Lep.), 596 (Misc. Dis.)
Taylor, J., 317 (Leish.)
Taylor, R. M., with Waddell, 33 (Y.F.)
Tchernomoretz, I., 539 (Leish.)
——, with Adler & Ber, 114 (Leish.)
Teixeira, Q. de A., 77 (Reports, etc.)
Tenen, M. M., with Iams & Flanagan, 587 (Der.)
Tennent, D. M., with Edison & Silber, 363 (Def. Dis.)
Terracini, E., with Romaña, 110, (110) (Tryp.)
Thangavelu, M., with Reddy, 495 (Lab.)
van Thiel, P. H., with Liem, 719 (Tryp.)
Thomas, C., with Drouet, Herbeuval & Faivre, 467 (Hel.)
Thomas, C. R., 195 (Rab.)
Thompson, A. W. S., 709 (Mal.)
Thompson, C. M., with McDaniel & White, 1060 (Def. Dis.)
Thompson, J. H., 98 (Mal.)
——, with Rifkin, 353 (Hel.)
Thompson, K. J., Rifkin & Zarrow, M., 468 (Hel.)
Thomsen, M., with Larsen, 1194 (Ent.)
Thomson, K. J., with Freund, Sommer, Walter & Schenkein, 311 (Mal.)
Thomson, R. C. M., 93 (Mal.)
Thonnard-Neumann, E., 520 (Mal.)
Tierney, N. A., 920 (Typh.)
Tilden, I. L., 455 (Lep.)
——, with Arnold, 452 (Lep.)
Tinkham, E. R., 671 (Vms.)
Tisseuil, J., (355) (Hel.)
Titman, W. L., with Cullinan, Kekwick & Watts, 942 (Def. Dis.)
Todd, A. R., with Hull, Lovell, Openshaw & Payman, (822) (Mal.)
Toledo, S. de A., (1182) (Oph.)
Tomlinson, W. J., 373 (Haem.)
Tompsett, R. R. & Kauer, G. L., 45 (Ys.)
Tonkin, I. M. & Work, T. S., 1012 (Mal.)
Tong-That-Tung, with Meyer-May, Ho-Dac-Di, Vu-Dinh-Tung & Dinh-Van-Thang, 87 (B.R.)
Toomey, A. G., 1006 (Mal.)
Topping, N. H., 20 *bis*, 31, 212 (Typh.)
——, Bengston, I. A. & Henderson, R. G., 20 (Typh.)
——, with —— & ——, 19 (Typh.)
——, —— & Shear, M. J., 18 (Typh.)
——, with Henderson, 20 (Typh.)
——, —— & Bengtson, I. A., 21 (Typh.)
——, with Irons, Shepard & Cox, 924 (Typh.)
—— & Shepard, C. C., 910, 911 (Typh.)
——, with ——, 21, 735 (Typh.)
Torrealba, F. J., 831 (Tryp.)
——, with Dias, 109 (Tryp.)
Torres, C. M. & Pinto, C., 658, 1156 (Hel.)
Tottey, M. M., with Maegraith, Adams, King, Rigby & Sladden, 821 (Mal.)
——, with Spinks, 398 *bis*, 400, 707 (Mal.)
——, with —— & Maegraith, (707) (Mal.)
Toullec, F., (595) (Misc. Dis.)
Toumanoff, C., 873 (B.R.)
Townshend, R. H., with Adams & King, 400 (Mal.)
——, with ——, Maegraith, King, Davey & Havard, 402 (Mal.)
——, with Maegraith, Adams, King, Davey & Havard, 402 (Mal.)
Trager, W., Bang, F. B. & Hairston, N. G., 821 (Mal.)
Traub, R., with Mackie, Davis, Fuller, Knapp, Steinacker, Stager, Jellison, Millspaugh, Austrian, Bell, Kohls, Wei Hsi & Girsham, 917 (Typh.)
Travassos, J. & Vallejo-Freire, A., 642 (Typh.)
Travis, B. V., with Gahan & Lindquist, 99 (Mal.)
——, with ——, Morton & Lindquist, 99 (Mal.)
——, with Lindquist, Madden & Husman, 795 (Ent.)
——, with ——, ——, Schroeder & Jones, 159 (Ent.)
Tredway, J. B., with Harvey & Kuffler, 490 (Misc. Dis.)
Trembley, H. L., with Coatney & Cooper, 629 (Mal.)
Trenchard, H. J., 1073 (Misc. Dis.)
Trespalacios, F. & González Prendes, M. A., 934 (Lep.)
Trim, A. R., with Alexander, 569 (Hel.)
Trinder, P., with Black & Fourman, 667, 1161 (Sp.)
Troitsky, N. V., 745 *bis* (R.F.)
Trout, C. L., 341 (Lep.)
Trowell, H. C., 684 (Reports, etc.), 1062 (Def. Dis.)
—— & Muwazi, E. M. K., 143, 363 (Def. Dis.)
Trussell, M., with Johnson & Jahn, 67 (Prot.)
Trussell, R. E. & Johnson, G., (254) (Prot.)
Trypanosomiasis Committee of Southern Rhodesia, 907 (Tryp.)
Tshinaev, P. P., 1077 (Ent.)
Tsuchiya, H., 332 (Am.)
—— & Kenamore, B., 743 (Am.)
Tubangui, M. A., 852 (Hel.)
Tucker, H. A., 1158 (Hel.)
——, with Kean, 1189 (Misc. Dis.)
——, with —— & Miller, 597 (Misc. Dis.)
Tucker, W. A. L., 561 (R.F.)
Tuey, G. A. P., (822) (Mal.)
——, with Curd, Davis, Owen & Rose, (1110) (Mal.)
Tumulty, P. A., Nichols, E., Singewald, M. L. & Lidz, T., 819 (Mal.)
Tupikova, N. A., with Gellhorn, van Dyke & Pyles, 722 (Leish.)
Tupinambá, A. A., with Martins & Versiani, 718 *bis* (Tryp.)
Turner, E. L., with Bent, Holloway & Cuff, 253 (Misc. Dis.)
Twining, H. E., Dixon, H. M. & Weidman, F. D., 674 (Der.)
Tye, M., 598 (Misc. Dis.)

U.

United States Nav. Med. Bull., 440 (Chl.)
United States Navy, (502) (Reports, etc.)
U.S. Pub. Health Service, 18 (Typh.)

Unsworth, K., with Bertram & Gordon, 1197 (Ent.)
—— & Gordon, R. M., 1195 (Ent.)
Unti, O., with Fonseca, (905) (Mal.)
Upholt, W. M., with Arnold & Ferguson, 793 (Ent.)
——, Gaines, T. B., Simmons, S. W. & Arnold, E. H., 794 (Ent.)
Upton, R. G., 649 (Pl.)

V.

Vaizey, J. M., 959 (Misc. Dis.)
Vallejo-Freire, A., with Travassos, 642 (Typh.)
van den Berghe, L., 491 (Misc. Dis.)
van den Ende, M., with Buckland, Dudgeon, Edward, Henderson-Begg, MacCallum, Niven & Rowlands, with Bargmann, Curtis & Shepherd, 436 (Typh.)
——, Locket, S., Hargreaves, W. H., Niven, J. & Lennhoff, L., 918 (Typh.)
Van der Kuyp, E., with Swellengrebel, 267 (B.R.)
Vanderplank, F. L., 494 (Ent.)
Van der Sar, A., 861 (Misc. Dis.)
Vanderscheer, J., with Wolfe, Clancy & Cox, 735 (Typh.)
Van der Walt, S. J. & Steyn, D. G., 960 (Misc. Dis.)
Vanni, V., 264 (B.R.)
Varay, A., with Hillemand, Debray & Duguet, 658 (Hel.)
Varela, G., 672 (Der.)
——, with Bustamante & Ortiz Mariotte, 1134 (Typh.)
——, with Mazzotti, 1135 (Typh.)
Vargas, L., 905 (Mal.)
Varley, F. M. & Weedon, F. R., 210 (Typh.)
Varnadoe, N. B., with Rigdon, 630 (Mal.)
Vaucel, 605 (Reports, etc.)
Vaughn, A. M., Howser, J. W. & Shropshear, G., (157) (Misc. Dis.)
Veeraraghavan, N., 194 (Rab.)
Veliath, G. D., with Menon, (297) (Mal.)
Venning, W. L., with Harrell, Wolff & Reinhart, 923 (Typh.)
Verdoorn, F., with Honig, 884 (B.R.)
Versiani, V., with Martins, (904) (Mal.)
——, —— & Sobrinho, O. P., 754 (Hel.)
——, with —— & Tupinambá, 718 *bis* (Tryp.)
Viel, B. & Romero, H., 545 (Typh.)
Villasana, A., with Martínez Báez, 747 (R.F.)
de Villiers, V. M., 498 (Reports, etc.)
Vilter, C. F., with Blankenhorn, Scheinker, & Austin, 945 (Def. Dis.)
——, with Spies, Cline & Frommeyer, 772 (Haem.)
——, —— & Koch, M. B., 375 (Haem.)
——, with ——, —— & Caldwell, 375 (Haem.)
Vincent, H., 250 (Ulc.)
Vincke, I., with Parent, M., 307 (Mal.)
——, ——, Berteaux, M. & de Meulenaere, J., 291 (Mal.)
Violle, H. & Calamet, L., (730) (Typh.)
Vishnievskaya, S. M., 565 (Hel.)
Viswanathan, D. K., 285 (Mal.)
Viswanathan, R., 252 (Misc. Dis.)
Vitlinskaya, I. A., with Leitman, 1143 (Am.)
Vizcarrondo R., O., 719 (Tryp.)
Vollmer, H. & Liebig, H., 201 (Mal.)
Vos, J. J. T., with Brug, 382 (Prot.)
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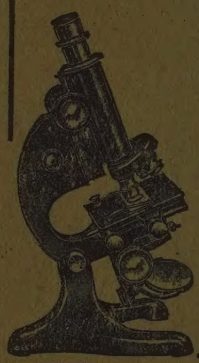

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